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FOR ENVIRONMENTAL QUALITY

ETHYLENE IN THE ENVIRONMENT: SCIENTIFIC CRITERIA FOR ASSESSING ITS EFFECTS ON ENVIRONMENTAL QUALITY

SUBCOMMITTEE ON AIR

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PANEL ON ETHYLENE IN THE ENVIRONMENT

Dr. D.M. Reid (Chairman)
Department of Biology
University of Calgary
Calgary, Alberta
T2N 1N4

Ms. M.G. Sheffer (Project Coordinator)
Environmental Secretariat
National Research Council of Canada
100 Sussex Drive
Ottawa, Ontario
K1A OR6

Dr. R.C. Pierce (Subcommittee Secretary)
Environmental Secretariat
National Research Council of Canada
100 Sussex Drive
Ottawa, Ontario
K1A OR6

Dr. D.F. Bezdicek
Department of Agronomy and Soils
Washington State University
Pullman, Washington
U.S.A. 99164

Dr. S.N. Linzon
Air Resources Branch
Ontario Ministry of the Environment
880 Bay Street, Suite 347
Toronto, Ontario
M5S 1Z8

Mr. T. Reuvers
Novacor Chemicals Ltd.
P.O. Box 5006
Red Deer, Alberta
T4N 6A1

Dr. M.S. Spencer
Department of Plant Science
University of Alberta
Edmonton, Alberta
T6G 2P5

Mr. F. Vena
Environmental Protection Service
351 St. Joseph Blvd.
Hull, Quebec
K1A 1C8

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SUMMARY

Physical-Chemical Properties (see Chapter 2)

Ethylene, a colorless gas with a sweet odor, is highly flammable, and may form explosive mixtures with air. The gas is only slightly soluble in water. Ethylene is adsorbed onto charcoal, rubber septa, etc. Some physical-chemical data for ethylene are tabulated in this chapter.

Sampling and Quantitative Analysis (see Chapter 3)

Standard methods of sampling air are appropriate for ethylene sampling, but require some modification. Care must be taken to avoid use of materials (such as rubber or plastic) that might adsorb or release ethylene, to avoid wound ethylene production, and to avoid gasoline-powered motors which produce large quantities of the gas. Methods similar to those used for sampling air can be used to estimate the ethylene content of the soil atmosphere. Ethylene in liquids can be collected by vacuum extraction or by stripping the ethylene from solution with the carrier gas of a gas chromatograph. Endogenous ethylene from tissues can be collected by vacuum extraction techniques. Care must be taken to avoid increased ethylene production resulting from tissue excision.

The concentration of ethylene in a sample is best estimated by gas chromatography using a flame ionization or a photoionization detector. If the sample contains a sufficiently high concentration of ethylene, quantitation is achieved by direct injection of a small air sample onto the gas chromatograph. Bigger sample volumes produce peaks with poor resolution. At lower concentrations of the gas, the ethylene must first be removed, trapped and concentrated from a large air sample, and later liberated for gas chromatographic analysis. A laser system that detects ethylene has also been devised.

It can be useful to have ethylene-free air available. This can be prepared by passing air over heated platinized asbestos or through a column packed with an ethylene absorbant.

Radioactively labelled ethylene is often used in biochemical studies of ethylene action and metabolism. [^{14}C]-ethylene, when purchased from commercial sources, may contain impurities; even after gas chromatographic purification, impurities can be detected as a result of radiation decomposition. Extreme caution must therefore be taken when using labelled ethylene in biological studies.

Sources and Emissions

Natural

Soil Microorganisms (see Section 4.1.1)

Soil is both a producer and consumer of ethylene. Most studies show that the major source of ethylene in soil is of microbial origin, although there has been debate as to the major producers. Evidence has been found supporting its production by facultative anaerobes, by several fungal species, and by spore-forming anaerobic bacteria. Methionine has been suggested as an ethylene precursor for fungi and for several gram-negative bacteria, but it does not appear to act as a precursor in Penicillium spp.

The evidence now appears to support the hypothesis that fungi are the dominant producers of soil ethylene, especially with regard to studies in pure culture. However, it is quite possible that two or more microorganisms co-metabolize precursors to produce ethylene in soil. Further studies are justified in view of the potential impact of ethylene on other microorganisms and plants.

The biosynthesis of ethylene by microorganisms has been studied in various fungi, bacteria and yeasts. Differences in ethylene biosynthesis are based on such parameters as (i) precursors (including acetate, glucose, glutamate and methionine); (ii) culture conditions; (iii) biosynthetic pathways and their regulatory mechanisms; and (iv) whether ethylene is produced enzymatically, cellularly or extracellularly in the medium.

Several factors have been found to influence ethylene production in soil. For example, temperature increases between 5 and 35°C can increase ethylene production, whereas autoclaving above 120°C prevents ethylene production. Soil drying also appears to enhance ethylene production, although this may be due to a reduction in ethylene decomposition.

Ethylene production also appears to increase in soils higher in organic matter, including barley and wheat straw, phenolic acids, animal wastes, lime, chitin and cellulose. Some fungicides also increase the ethylene concentration in the soil atmosphere, possibly because of an increase in available substrate for ethylene producers after cell lysis.

Ethylene production has been shown to increase as oxygen levels decrease in relation to the level of soil flooding. Ethylene accumulates more in soil under reduced conditions than under oxidized conditions; this possibly reflects the greater tendency for ethylene to degrade under more oxidized conditions. With respect to soil pH, ethylene production is expected to be optimum in the neutral pH range, coinciding with optimal soil microbial activity. A negative relation

between soil nitrate level and ethylene production has been observed, possibly because the nitrate levels keep the redox potential at a value above which ethylene production begins. However, other studies have not shown any relationship between ethylene production and soil nitrate level.

Plants (see Section 4.1.2)

Ethylene is produced by most, if not all, plants. The rate of normal ethylene production varies with the kind of plant, with the part of the plant, and with the stage of development. For most biological tissues, ethylene production ranges from 0.5 to 6 $\mu L.kg^{-1}.h^{-1}$ (see Table 2-1 for conversion factors), although large variations have been observed. Because of the limitations of commonly used methodology, there are at present few reliable data on ethylene production by plants.

A major biosynthetic pathway for ethylene in a number of higher plant tissues has been established, and involves the formation of 5'-methylthioadenosine (MTA) and 1-aminocyclopropane-1-carboxylic acid (ACC) from S-adenosylmethionine (SAM) and the recycling of methionine. Increased ethylene synthesis can result from the enhancement of the activity of ACC synthase, whereas ethylene production can be inhibited if the conversion of ACC to ethylene is inhibited or if ACC is irreversibly converted to a different compound. Recent work shows that polyamines also arise from SAM, and suggests that the formation of ethylene and polyamines may be competitive under certain conditions.

Some evidence exists for other pathways for the biosynthesis of ethylene by higher plants.

Humans and Other Animals (see Section 4.1.3)

Ethylene appears to be produced by humans, rats and mice; both exhalation of ethylene by humans, rats and mice and the production of ethylene by subcellular fractions from rat liver have been observed. The extent of experimentation is, however, extremely slim. There has been little experimentation on the biosynthesis of ethylene in animals, or its control.

Anthropogenic (see Section 4.2)

In Canada, information is fragmentary on the quantities of ethylene emitted from anthropogenic sources. The emission estimates presented in this section are to be treated as preliminary, since they rely principally on estimates of emissions of total volatile organic compounds coupled with available data on the ethylene fraction of those emissions extracted from published information.

The emissions of ethylene in Canada in 1980 from anthropogenic sources amounted to about 121 100 Mg (1 Mg = 1 tonne). This total was made up of 84 300 Mg (70%) from transportation sources, 25 000 Mg (21%) from the production of ethylene and ethylene-derived petrochemicals, 8840 Mg (7%) from fuel combustion in stationary sources, and the remaining 1870 Mg (2%) from other miscellaneous sources.

Gasoline-powered motor vehicles (on-road plus off-road) are the major source of ethylene emissions, contributing about 68 800 Mg or slightly over 50% of the anthropogenic emissions of ethylene in Canada in 1980.

Levels in the Environment (see Chapter 5)

Few measurements have been made of ambient ethylene concentrations in Canadian locales. In general, low ethylene levels in air are associated with rural areas and small towns, whereas high levels are associated with cities and proximity to industrial and high traffic areas. In addition to location, time of day, time of year, and meteorological conditions also influence the atmospheric concentration of ethylene. Typical values range from less than 15 $\mu g.m^{-3}$ for a natural wilderness area away from automobiles and industry to 50-60 $\mu g.m^{-3}$ for cities like Calgary and Los Angeles, and up to 800 $\mu g.m^{-3}$ in the center of Washington, D.C. Concentrations of ethylene in indoor air as a result of tobacco smoking have not been measured.

High concentrations of ethylene appear to be unlikely in well aerated soils. The gas phase of anaerobic soils has been found to contain 12 $mg.m^{-3}$ ethylene. Waterlogged soils contained higher concentrations of ethylene, between 15 and 20 $mg.m^{-3}$ in air at equilibrium with the soil water.

Ethylene has been measured at concentrations of less than 35 $nL.L^{-1}$ in several marine locations.

In plants, internal ethylene levels increase as the rate of ethylene production increases, and thus depend on many factors that affect the rate of production, e.g. organ and stage of development. In addition, exogenous ethylene causes the internal levels to increase. Internal concentrations range from 0.01 $mg.m^{-3}$ in bananas to 2900 $mg.m^{-3}$ in apples.

In humans, exhaled air was found to contain 8 $mg.m^{-3}$ ethylene, twice the content of the inhaled air.

Environmental Dynamics

Abiotic Fate (see Section 6.1)

Once released into the atmosphere, ethylene is dispersed by turbulent processes and may participate in two major photochemical reactions: reaction with ozone and reaction with the hydroxyl radical. The reaction of ozone with ethylene is considered to be bimolecular, with a rate constant of 1.8×10^{-18} $cm^3.s^{-1}$ per molecule, and leads to complex degradation pathways giving rise to such products as ozonides, aldehydes, carbon monoxide, carbon dioxide and acids. The initial reaction of the hydroxyl radical with ethylene is also considered to be bimolecular, having a rate constant of about 8.3×10^{-12} $cm^{-3}.s^{-1}$ per molecule; subsequent radical chain reactions lead to the formation of formaldehyde, nitrogen dioxide, and small amounts of formic acid.

At an ozone concentration of 1×10^{12} molecules per cubic centimetre, the rate of reaction of ethylene and ozone is about 1.8×10^{-6} s^{-1}; the residence time of ethylene in the presence of ozone would be about 6.5 d. Similarly, assuming a hydroxyl radical concentration of 1×10^{5} molecules per cubic centimetre, the residence time would be about 14 d. Ethylene appears to be one of the least photochemically reactive compounds of the vapor-phase, unsaturated, nonaromatic hydrocarbons.

Biotic Fate

Soil Microorganisms (see Section 6.2.1)

Little is known about the metabolism of ethylene by soil microorganisms, a significant process when one considers that one-half of the annual ethylene production in the United States is degraded microbiologically in the soil. The catabolic pathway of ethylene in Mycobacterium E20 is possibly the most well understood in soil microorganisms at this time. Ethylene is oxidized to ethylene oxide by an enzyme system utilizing a monooxygenase; the ethylene oxide does not yield ethylene glycol.

The consumption or decomposition of ethylene appears to be carried out by a group of aerobic organisms at a rate much higher than that of production. Decomposition is affected by such factors as autoclaving, soil moisture, O_2 and CO_2 concentrations, and glucose addition.

Plants (see Section 6.2.2)

It is assumed that the greater part of ethylene enters higher plants through the stomata; its ease of uptake would therefore depend on the number and size of the stomatal openings, as well as on the

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diffusion gradient in the plant. After uptake, ethylene should move readily through plant tissues by diffusive processes, its small size as well as its solubility in both lipophilic and aqueous systems facilitating its movement through air spaces and cell membranes. The ease of diffusion and the diffusion path for ethylene appear to depend on the tissue involved. Some of the gas can be removed from the plant by diffusion to the external atmosphere.

Several groups have examined the incorporation of ethylene into plant tissues and its metabolism. Early results claiming incorporation and metabolism were discounted, largely because of impurities in the radioactive ethylene used in the experiments. However, it now appears that ethylene can be metabolized in a number of plant tissues, although few generalizations can be made concerning the metabolic process. In Pisum sativum, for example, ethylene is metabolized to ethylene oxide which is then hydrolyzed to ethylene glycol. Carbon dioxide has also been observed to be a metabolite of ethylene in plants. However, some tissues do not appear to metabolize ethylene, and there are even discrepancies within a species of plant.

The physiological significance of ethylene metabolism in plant tissues is also not well understood. It has been suggested that ethylene metabolism may regulate endogenous ethylene levels, although several observations, including the lack of increase in amount of ethylene evolved from tissues when metabolism is inhibited, contradict this. An alternate suggestion, that ethylene metabolism occurs at the binding site as an integral part of the ethylene action mechanism, is gaining support, and leads to the hypothesis that there are two ethylene metabolizing systems in plant tissues: one produces ethylene oxide, and is inhibited by silver ions (Ag^+); the other yields CO_2, and is inhibited by CO_2. The ethylene oxide may be further metabolized to ethylene glycol in some tissues.

Humans and Other Animals (see Section 6.2.3)

Ethylene appears to be taken up rapidly from the lungs in humans and other animals. Although some of the compound or its metabolites may be retained by tissues, most is eliminated rapidly via exhalation. Urinary excretion also plays a role in ethylene elimination.

The results of early investigations implied that ethylene was not metabolized by humans and other animals. However, recent findings, including the exhalation of ethylene oxide by rats exposed to high concentrations of ethylene, the occurrence of hydroxyethylcysteine in the urine of mice exposed to ethylene, the similar ratios between alkylated cysteine and alkylated histidine in the hemoglobin of mice exposed to ethylene or to ethylene oxide, and the in vitro formation of ethylene oxide from ethylene by rat liver microsomes, all seem to suggest that ethylene is metabolized to ethylene oxide in animals,

possibly by a mixed function oxidase. The ethylene oxide may then be further metabolized to ethylene glycol, although this has not yet been observed in animals.

Several investigations of the uptake, retention, and elimination of ethylene by rats pretreated with chemicals that induce the hepatic mixed function oxidase system have also supported the contention that ethylene is metabolized to ethylene oxide. For example, ethylene metabolism appears to be stimulated by pretreatment with polychlorinated biphenyls (PCBs), resulting in increased tissue and excreta concentrations of radioactivity associated with less volatile metabolites of ethylene rather than with the parent compound.

Effects

Soil Microorganisms (see Section 7.1)

It has been suggested that ethylene is a fungistatic agent, inhibiting propagule growth by Sclerotium rolfsii and Helminthosporium sativum in soil. However, there has been little work that substantiates this finding, and it appears that the fungistatic effect of ethylene, if any, is limited to a small number of species.

Plants (see Section 7.2)

Ethylene is a unique air pollutant in that it also acts as a plant hormone, affecting a large number of physiological and biochemical processes. Although it cannot be assumed that because exogenously applied ethylene has a particular effect, endogenous ethylene controls this parameter of growth and development, the use of antagonists of ethylene biosynthesis and action can usually provide a definitive answer.

There have been relatively few field observations of damage to vegetation by ethylene from such sources as illuminating gas, industries utilizing or producing ethylene, greenhouses, and automobile exhaust. Fruit, flowers and nursery stock appear to be the most sensitive species in these situations.

Ethylene's effects on plants, tabulated in this section, include influences on dormancy, rate and orientation of cell expansion, rate of aging, osmotic swelling of motor cells, development and differentiation, and many specific biochemical processes. Ethylene can have both stimulatory and inhibitory effects on a particular process; this can perhaps be explained by a biphasic dose-response curve which can shift to the left or right depending on such factors as type or age of the tissue under study. It is, however, difficult to confirm this hypothesis, because (i) tiny quantities of ethylene may be required for growth and development; (ii) background concentrations of ethylene

differ from location to location; and (iii) some cells may be responsive to ethylene only for a short period, or one aspect of their growth may be inhibited and stimulated by ethylene at different stages of development.

Little work has been done on dose-response-time relationships for ethylene. In addition, many studies have used unnaturally high levels of ethylene. A general conclusion is that 0.01 $mg.m^{-3}$ is needed for a threshold effect, and 0.1-0.6 $mg.m^{-3}$ for a maximum effect. Many factors can modify these limits, including background ambient ethylene, the duration of exposure, the age and type of tissue, and the presence of interacting environmental parameters (e.g. injury, freezing, flooding, and other air pollutants such as ozone).

Because ethylene can act on such a diversity of biochemical, growth and differentiation parameters, it is reasonable to argue that the gas must be able to control some fundamental and central process. Apparently it is ethylene that is active, not some related form of compound, since ethylene is more potent than its precursors, chemicals that release ethylene, ethylene oxide, and other related compounds. Several avenues have been investigated regarding the molecular action of ethylene, including (i) the binding of ethylene to a metal at the site of action; (ii) the interaction of ethylene with membranes; (iii) the close relationship between ethylene metabolism and action; and (iv) the effect of ethylene on microtubule orientation. None of these approaches as yet has given us the definitive answer on how ethylene operates at the molecular level.

Because ethylene may be metabolized to ethylene oxide and ethylene glycol in certain plant species, the effects of these compounds on plant processes should also be examined. Little work has been performed in this area, although ethylene oxide is known to act synergistically with ethylene in the growth inhibition of peas, as well as amplifying other ethylene effects.

Humans and Other Animals (see Section 7.3)

Ethylene, at concentrations around 80%, was used as an anesthetic for many years. Since its use was discontinued, several workers have investigated the possibility that ethylene causes effects other than anesthesia and asphyxia in animals. Although some investigators found no effects on such gross physiological variables as weight gain, organ lesions and lung irritation, at the subcellular level ethylene has been observed to affect mitochondrial volume, the action of mitochondrial adenosine triphosphatase, and peroxidases.

In addition, recent data suggest that ethylene destroys the hepatic microsomal cytochrome P-450 monooxygenase in phenobarbital-pretreated rats. Severe degeneration and necrosis of the liver have been observed in rats pretreated with Aroclor 1254 and then exposed to

ethylene. Further experiments studying various enzymes involved in epoxide detoxification support the suggestion that pretreatment with chemicals inducing the hepatic mixed function oxidase system affected the rate or route of ethylene metabolism so that a toxic metabolite was produced; the metabolite was either not produced in the controls or was produced in insufficient quantities to elicit a hepatotoxic response.

Since it is quite likely that ethylene gives rise in vivo to ethylene oxide, an alkylating agent with established mutagenicity, carcinogenicity, and teratogenicity, and since it is not known whether tissue doses of ethylene oxide generated in vivo from inhaled ethylene exert the same effect as tissue doses obtained through inhalation of the epoxide, several groups have attempted to estimate the genetic risk to man associated with exposure to ethylene. It is possible that the effects of ethylene on humans and other animals resulting from metabolism to ethylene oxide are not too severe, because of such factors as (i) the low levels of ethylene to which animals are exposed; (ii) the rapid exhalation of ethylene; (iii) the low conversion of absorbed ethylene to ethylene oxide; and (iv) the detoxification of ethylene oxide in the liver. However, before any definitive conclusions can be drawn, much more work needs to be done.

RECOMMENDATIONS

There does not appear to be enough information available at the present time to determine if there is an ethylene pollution problem in Canada. It is particularly important to determine the concentrations of ethylene present in the environment together with the long-term effects of these concentrations on sensitive organisms. The Panel has therefore identified the following major areas, in order of priority, as those requiring research in order to fill gaps in knowledge regarding ethylene in the Canadian environment.

1. CONCENTRATIONS

In order to establish whether or not there is an ethylene pollution problem in Canada, it is essential to determine the national occurrence of ethylene in the Canadian environment. Although emission estimates have been presented in this document, they rely principally on estimates of emissions of total volatile organic compounds, and hence should be treated only as preliminary estimates. Since there are almost no published data on ethylene levels in Canada, it is recommended that:

(i) MEASUREMENTS BE MADE OF ETHYLENE CONCENTRATIONS IN URBAN AND RURAL AREAS, AND IN THE VICINITY OF INDUSTRIES, HIGHWAYS, AND VEGETATION. PARTICULAR ATTENTION SHOULD BE PAID TO CONCENTRATIONS IN THE VICINITY OF ROADS AND HIGHWAYS, SINCE GASOLINE-POWERED MOTOR VEHICLES ARE THE MAJOR SOURCE OF ETHYLENE EMISSIONS IN CANADA. CONCENTRATIONS OF ETHYLENE IN THE INDOOR ENVIRONMENT SHOULD ALSO BE MEASURED.

Because it is expected that ethylene levels will vary not only spatially, but also temporally, it is recommended that:

(ii) MONITORING PROGRAMS BE DESIGNED SO THAT DIURNAL, SEASONAL AND YEARLY VARIATIONS IN THE ABOVE MEASUREMENTS CAN BE DETERMINED.

Because ethylene is able to cause damage to vegetation at extremely low concentrations, it is recommended that:

(iii) A MORE SENSITIVE, ETHYLENE-SPECIFIC METHOD OF MONITORING AND RECORDING BE DEVELOPED WITH A DETECTION LIMIT AT THE 1 $\mu g.m^{-3}$ LEVEL.

2. EFFECTS

It is well known that ethylene can cause gross visible damage to greenhouse crops as well as to sensitive flowers, such as orchids. However, most dose-response work was performed fifty years ago using questionable methodology as well as ethylene concentrations much higher than those commonly encountered in the Canadian environment. In

addition, there is almost no work examining the effect of exposure time on dose-response relationships, nor are there data on modes of action and physiological responses for a sufficiently wide range of plant species, especially those found growing in Canada. Finally, there is almost no information on the possible effects of ethylene on animals, including humans. Since air quality objectives are based on such data, it is recommended that:

(i) RESEARCH BE UNDERTAKEN TO DETERMINE PRECISE DOSE-RESPONSE-TIME RELATIONSHIPS IN PLANTS AND MICROBIAL SPECIES OF IMPORTANCE IN CANADIAN FORESTRY AND AGRICULTURE/HORTICULTURE. PARTICULARLY SENSITIVE CANADIAN SPECIES SHOULD BE IDENTIFIED. WHEREVER POSSIBLE, FIELD CONDITIONS SHOULD BE SIMULATED WITH RESPECT TO ETHYLENE LEVELS AND SUCH ENVIRONMENTAL PARAMETERS AS TEMPERATURE, NUTRIENT AND WATER SUPPLY, WIND SPEED AND IRRADIANCE. ATTENTION SHOULD BE PAID TO THOSE PERIODS OF PLANT GROWTH AND DEVELOPMENT THAT MIGHT BE PARTICULARLY SENSITIVE TO ETHYLENE.

(ii) ATTENTION SHOULD BE PAID TO ANY POTENTIAL EFFECTS ON HUMAN OR ANIMAL HEALTH OF CONTINUOUS EXPOSURES TO LOW LEVELS OF ETHYLENE AND OF INTERMITTENT EXPOSURES TO RELATIVELY HIGH CONCENTRATIONS.

(iii) ALTHOUGH THE DETERMINATION OF DOSE-RESPONSE-TIME RELATIONSHIPS FOR SENSITIVE CANADIAN SPECIES IS OF PARAMOUNT IMPORTANCE, SEVERAL ADDITIONAL STUDIES COULD BE PERFORMED WHICH WILL AID IN THE UNDERSTANDING OF ETHYLENE ACTION:

- THE ROUTES BY WHICH ETHYLENE ENTERS, IS TRANSPORTED, AND IS ELIMINATED FROM PLANT, ANIMAL, AND MICROBIAL ORGANISMS, AND THE RATES OF THESE PROCESSES, SHOULD BE DETERMINED.

- STUDIES SHOULD BE PERFORMED ON THE RATES AND MODES OF ETHYLENE METABOLISM IN PLANT, ANIMAL AND MICROBIAL ORGANISMS. ATTENTION SHOULD BE PAID TO THE TYPES OF METABOLITES FORMED, THEIR FATES AND THEIR TOXICITIES.

- THE MOVEMENT OF ETHYLENE AND ITS PRECURSORS AMONG DIFFERENT SPECIES SHOULD BE EXAMINED. EXAMPLES OF THESE INTERACTIONS INCLUDE TRANSFERS AMONG DIFFERENT SPECIES OF SOIL MICROORGANISMS AND AMONG SOIL MICROORGANISMS AND PLANTS.

- MODES OF ACTION OF ETHYLENE AND ITS METABOLITES AT THE BIOCHEMICAL LEVEL AND THE PHYSIOLOGICAL MANIFESTATIONS OF THESE ETHYLENE-INDUCED CHANGES SHOULD BE INVESTIGATED IN PLANTS AND ANIMALS.

3. SOURCES/SINKS/TRANSPORT/TRANSFORMATIONS

In Canada, information is fragmentary on the quantities of ethylene emissions from anthropogenic and natural sources, in particular from such diverse sources as soil, plants, aqueous systems, agricultural burning and forest fires. To adequately predict the fate of ethylene in the Canadian environment, various sources of and sinks for ethylene need to be identified, along with transport and transformation mechanisms. It is therefore recommended that:

(i) RESEARCH BE PERFORMED TO FURTHER QUANTIFY THE CANADIAN EMISSIONS OF ETHYLENE FROM ANTHROPOGENIC AND NATURAL SOURCES, IN PARTICULAR EMISSIONS FROM TRANSPORTATION, INDUSTRIAL PROCESSES, AGRICULTURAL AND FORESTRY APPLICATIONS, FUEL COMBUSTION, SOILS AND VEGETATION.

(ii) RESEARCH BE CARRIED OUT TO INCREASE OUR KNOWLEDGE ABOUT THE ATMOSPHERIC TRANSPORT AND BIOTIC AND ABIOTIC FATES OF ETHYLENE UNDER TYPICAL CANADIAN CONDITIONS.

4. INTERACTIONS

It is likely that any receptor in Canada will be exposed to a complex mixture of air pollutants rather than to ethylene alone. It is therefore important that the effects of ethylene in conjunction with such common air pollutants as sulfur dioxide, nitrogen oxides, formaldehyde and ozone be examined. In particular, because of the known interactions between carbon dioxide and ethylene action in plant tissues, it is important to determine the influence of increased levels of CO_2 in the atmosphere on the effects of ethylene on plant receptors. In addition, the influence of such environmental variables as temperature, water supply, and plant pathogens on the actions of ethylene should be investigated. It is therefore recommended that:

INTERACTIONS BETWEEN ETHYLENE AND OTHER ENVIRONMENTAL STRESSES AND VARIABLES BE EXAMINED WITH RESPECT TO THEIR EFFECTS ON RECEPTORS.

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1.0 INTRODUCTION

In the past, ethylene as an air pollutant has been studied much less than such toxicants as ozone and sulfur dioxide for several reasons, the foremost being the relatively limited economic consequences of ethylene pollution and the lack of demonstrated adverse effects on human health.

Concerns have recently been expressed, however, regarding the potential environmental and health effects that may result from the release of ethylene to the Canadian atmosphere. Some emissions of ethylene, particularly industrial and automobile exhausts, have been observed to affect plants in the vicinity of the sources. In addition, it now appears that ethylene has the potential to adversely affect human health. As a result of these concerns, the Subcommittee on Air of the NRCC Associate Committee on Scientific Criteria for Environmental Quality requested that a Panel be assembled to prepare a criteria document on the topic of ethylene in the environment, with special emphasis placed on Canadian emissions of ethylene and atmospheric conditions. This document, the end result of the Panel's efforts, assesses the current analytical methodology for ethylene, describes natural and anthropogenic sources of ethylene in Canada and ethylene levels in the environment, details the abiotic fate of ethylene in the atmosphere and its biotic fate in microorganisms, plants, and animals, and discusses the known dose-response relationships between ethylene and these same organisms. In addition, the report identifies gaps in knowledge and makes recommendations for research to fill those gaps.

2.0 PHYSICAL-CHEMICAL PROPERTIES

Ethylene is a colorless gas with a sweet odor. It is highly flammable, and burns with a luminous flame. Air and ethylene may form explosive mixtures. Ethylene has exploded spontaneously when mixed with chlorine and exposed to sunlight (Miller 1969).

Electrophilic agents such as strong acids, halogens, and oxidizing agents are reactive with ethylene. Ethylene reacts with nitric oxide and ozone in atmospheric photooxidation (Abeles 1973).

Ethylene is only slightly soluble in water (Miller 1969). It is marginally lighter than air, but vapor clouds tend to hug the ground and spread, rather than lift and disperse. Its molecular diffusivity in air at standard temperature and pressure is 0.137 $cm^2.s^{-1}$ (Pritchard and Currie 1982).

Particles with large surface areas, such as charcoal and silica gel, readily adsorb ethylene. Several researchers have observed the ability of ethylene to adsorb onto, and subsequently diffuse from, such materials as rubber septa and tubing (e.g. Kavanagh and Postgate 1970).

Some physical-chemical data for ethylene are summarized in Table 2-1.

Table 2-1. Physical-chemical data for ethylene (from Stahl 1969).

Synonyms	Ethene, elayl, olefiant gas, etherin
Formula	C_2H_4
Molecular weight	28.05
Melting point	-169.2°C
Boiling point	-103.8°C
Vapor density (air = 1)	0.978 (0°C)
Vapor pressure	4040 kPa (-15°C)
Flammability range (in air)	Lower: 2.7% Upper: 36%
Autoignition temperature	450°C
Solubility in water	0.015 g $(100\ g)^{-1}$ (20°C)
Other solubilities	Decomposes slightly in ethanol, acetone and benzene; soluble in diethyl ether
Log_{10} octanol/water partition coefficient	1.13
Odor threshold	300-800 $mg.m^{-3}$[α]

[α] At 25°C, 1 $mg.m^{-3}$ ethylene is approximately equivalent to 0.87 ppm ethylene. Both sets of units will be used throughout this document. In addition, where the quantity of ethylene has been originally expressed in microlitres, the conversion to nanomoles will be made, assuming an ideal gas, 1 atmosphere pressure, and 25°C (1 µL = 40.8 nmol ethylene).

3.0 SAMPLING AND QUANTITATIVE ANALYSIS

3.1 SAMPLING TECHNIQUES

3.1.1 ETHYLENE FROM AIR

Standard methods of sampling air as used in any air pollution project are appropriate (Cheremisinoff and Morresi 1978), but require some modification. Automated methods of ethylene sampling and analysis are also available (Dilley et al. 1969; Lougheed et al. 1969; Smith and Dowdell 1973; Watada and Massie 1981).

Since many industrial, agricultural and natural processes evolve ethylene (see Chapter 4), care must be exercised to take this into account. For instance, extensive trampling of plants can cause the liberation of remarkably high levels of ethylene. This is due to the production of ethylene from wounded tissue (Abeles 1973). In that situation the increase in ethylene may take only a few minutes but depends upon the type of tissue and the extent of the damage. Gasoline-powered motors, when running, also produce large quantities of the gas (see Section 4.2.1). Thus, one should be aware of sudden changes of wind speed and direction and other conditions at the sampling site.

Generally with the sampling equipment one should avoid using any materials, such as petroleum grease, that tend to either adsorb or release ethylene. Some plastics, when heated or exposed to light, can liberate ethylene (Jacobsen and McGlasson 1970). Teflon air sampling bags are quite suitable for the storage of ethylene samples for a few weeks. "Plastipak" disposable syringes are cheap, airtight and sufficiently accurate; however, they do retain high levels of ethylene. Gas-tight syringes with Teflon plunger tips are excellent.

3.1.2 ETHYLENE FROM SOIL

Similar methods to those used for sampling ethylene in air can be used in the estimation of the ethylene content of the soil atmosphere (Dowdell et al. 1972; Smith and Dowdell 1973; Smith 1977). With respect to measuring the production of ethylene by soil microorganisms, it should be pointed out that data from laboratory studies are often not directly applicable to field conditions. Measurement of ethylene in closed containers in the laboratory does not provide a true indication of production, but rather of accumulation, since ethylene decomposers are more significant under the closed atmospheric conditions. In addition, production would not proceed normally under these conditions because of depletion of oxygen and accumulation of carbon dioxide and ethylene.

3.1.3 ETHYLENE FROM TISSUES AND LIQUIDS

It is possible to collect ethylene in liquids by vacuum extraction or by stripping the ethylene from solution with the carrier gas of a gas chromatograph (Bassi et al. 1981). Endogenous ethylene from tissues can be collected by vacuum extraction techniques (Ward et al. 1978; Fabijan et al. 1981). The method of Beyer and Morgan (1970), in which tissues are held under the surface of a saturated solution of $(NH_4)_2SO_4$ during the extraction, can lead to inaccuracies in the estimation of the ethylene level because some ethylene may remain in the solution from previous sampling. In all these techniques for removal of ethylene from tissues, the tissue needs to be excised before being placed under vacuum. This can lead to erroneous data because of changes in rates of ethylene production resulting from the wounding of tissue and other alterations in metabolism (Bassi and Spencer 1983a, b).

The rates of production of ethylene from detached or intact tissues kept in closed containers can also be monitored. However, these techniques involve many problems. In closed systems there is a continually changing gaseous atmosphere. Changes in concentrations of carbon dioxide and other gaseous metabolites influence metabolism, including ethylene production (Dhawan et al. 1981; Bassi and Spencer 1982). Also, lowering the oxygen levels in closed systems may suppress ethylene production (Pratt and Biale 1944; Burg and Thimann 1959; Mapson and Robinson 1966; Wright 1977). Recently (Bassi and Spencer 1979), a method has become available for the determination of ethylene production by intact plants in an open, flow-through system, using air purified of hydrocarbons (Eastwell et al. 1978, 1981). In this apparatus, it is possible as well to separate ethylene production by shoots of the intact plant from that by roots or soil, to provide light for the plant, and to maintain desired temperatures. A flow-through system without temperature control or air free of hydrocarbons has been reported by De Greef and De Proft (1978).

Accurate measurement of the rate of ethylene production by living organisms requires extreme caution to avoid erroneous data. The apparatus should be made of inert materials, the air supplied should be purified, and changes in the gaseous atmosphere should be avoided.

3.2 QUANTITATIVE ANALYSIS

3.2.1 RANGE OF TECHNIQUES AVAILABLE

After the gas sample containing the ethylene has been obtained, the level of ethylene must be estimated. Quite a few techniques have been developed over the years for the analysis of ethylene, and most of these have been described by Abeles (1973). The level of ethylene is best estimated by a gas chromatograph fitted with either a flame ionization detector (FID) (Abeles 1973) or a photoionization detector (PID) (Bassi and Spencer 1985). A PID can

analyze ethylene at concentrations near a fraction of 1 $pL.mL^{-1}$ (ppb), using a 1-mL sample. For FID, 10 $pL.mL^{-1}$ (ppb) in a 5-mL sample is the limit of sensitivity. None of the other methods of quantitative analysis of ethylene is as quick, convenient or accurate as GC. Biological assay methods are unreliable, variable and of limited use for quantitative work. Nevertheless, they are cheap and can be useful as indicators that a fumigation episode has occurred.

3.2.2 GAS CHROMATOGRAPHY

If the concentration in air is high enough, one can directly inject the sample, without preconcentration, into the gas chromatograph. Large samples (>5 mL for FID, >1 mL for PID) produce peaks with poor resolution. With lower concentrations of the gas one must resort to the methods described in Section 3.2.3.

The GC-FID or PID methods have the advantage that they are rapid and accurate. If the gas chromatograph is linked to a microprocessor integrator, the time from injection to detection and estimation of the quantity of ethylene in the sample might be as short as 0.5 to 2 min, depending upon column type and temperature.

A typical setup for GC ethylene analysis would use 0.32-cm OD, 183-cm-long glass or stainless steel columns filled with any of the following - silica gel, alumina F, Poropak N or Q, Spherocarb (all 80-100 mesh) - and column temperatures of 60-70°C for the Poropaks, 140°C for Spherocarb, and 110°C for the others. Nitrogen is a good carrier gas (flow rate of 30 $mL.min^{-1}$). A number of other suitable columns and conditions are given by Ward *et al.* (1978) and Abeles (1973). The column used should give a clear separation of ethylene from acetylene, ethane, methane, acetaldehyde and ethanol, since these can be commonly found in tissues and air. It is wise to test one's samples on at least two different columns with different retention times for these gases. Large amounts of water vapor contaminating samples can reduce the efficiency of the column; however, this problem can be easily overcome by raising the column temperature by 40 or 50 C° for 10-15 min.

Sometimes, since ambient air will probably contain some ethylene, it can be useful to have available ethylene-free air. This can be achieved by passing ethylene-contaminated air over heated platinized asbestos (Eastwell *et al.* 1978, 1981). Eastwell *et al.* (1978) evaluated the efficiencies of 22 methods for the removal of ethylene (as well as ethane and methane) from air. The effectiveness of Purafil (composed of inert beads covered with potassium permanganate) in removing ethylene depends upon the dimensions of the column, the amount of absorbant, the gas flow rate, and the concentration of ethylene and other contaminating gases. It must be replaced periodically, whereas the catalytic oxidizers, such as platinized asbestos, have potentially infinite life.

A laser system that detects ethylene and other air pollutants has been devised (Patty et al. 1974; Menzies and Shumate 1976; Shumate 1976). As pointed out by Menzies and Shumate (1976), the laser system is a little cumbersome but has the advantage of being able to measure air pollutant levels over large areas from one location. The method appears to be sensitive enough to show significant diurnal variations in the concentration of ethylene in the air near roads.

3.2.3 METHODS FOR LOW LEVELS OF ETHYLENE IN AIR

The basic method for measuring low levels of ethylene is to take a large sample of air and remove, trap and concentrate the ethylene from this sample. One method is to trap the hydrocarbons by bubbling the air through a solution of mercuric perchlorate. This solution specifically absorbs hydrocarbons and thus concentrates them. These gases (including the ethylene) can then be liberated into a small volume by adding lithium chloride (Young et al. 1952; Abeles 1973). This method has the disadvantages that mercuric perchlorate is corrosive, poisonous and explosive. Also, the flow rate and bubble size are critical for the quantitative absorption of ethylene (Chandra and Spencer 1963a). The technique of Stinson and Spencer (1969), Eastwell et al. (1978) and De Greef et al. (1976) is better. In this method air is passed into a cooled pre-column trap (containing silica gel or a Poropak) kept at approximately -80°C by a dry ice - acetone pack (allowing the bulk of the air to pass freely through, but trapping the hydrocarbons). The hydrocarbons are then released by heating the trap to approximately 100°C for 5 min, and the now small volume of gases can be passed directly into the gas chromatograph. The trap can, if desired, be plumbed directly into the carrier gas line of the gas chromatograph. Since the collection system is subject to large variations in temperature during collection and release of ethylene, care should be taken to ensure that the system remains airtight during the pressure changes created by changes in temperature. These collection traps are relatively inexpensive (~$100-200) compared with ready-made units (several thousand dollars) that appear to have the same function (e.g. Nutech 320 Thermal Desorption System).

Other ethylene traps using liquid nitrogen are described by Abeles (1973). However, at liquid nitrogen temperatures (-190°C), oxygen also tends to condense out of the airstream, making it difficult to maintain a steady flow through the trap (Eastwell et al. 1978) and to achieve quantitative collections. None of these traps is specific for ethylene; thus, the use of a good GC analytical column between the trap and the FID is needed to separate the ethylene from other hydrocarbons or olefins.

3.2.4 [^{14}C]-ETHYLENE

In biochemical studies of ethylene action and metabolism it can be useful to employ radioactively labelled ethylene. [^{14}C]-C_2H_4 is

often used and when purchased from commercial sources may contain impurities (including acetylene), many or most of which derive from radioactive decomposition (Tolbert and Lemmon 1955). Beyer (1975d) showed that shortly after gas chromatographic purification, impurities were again detected as a result of radioactive decomposition. Storage of highly purified $[^{14}C]$-C_2H_4 was also unwise. Beyer (1975d) states that, "in view of the instability of $[^{14}C]$-C_2H_4 and the incorporation of its radiolysis products into plant products, extreme caution must be taken when using labelled $[^{14}C]$-C_2H_4 in biological studies." A procedure for the purification of $[^{14}C]$-C_2H_4 is described by Beyer (1975d).

4.0 SOURCES AND EMISSIONS

4.1 NATURAL

Ethylene can be produced by several natural sources, including soil microorganisms, plants, and humans and other animals.

4.1.1 SOIL MICROORGANISMS

4.1.1.1 Producers

Soil is both a producer and consumer of ethylene. Most studies show that the major source of ethylene in soil is microbial, although there has been debate as to the major producer(s). Smith and Restall (1971), the first to suggest that ethylene is produced by facultative anaerobes in soil, found concentrations of 23 $mg.m^{-3}$ in the soil atmosphere. They pointed out that ethylene production in soil reached a maximum before methane production in soil, suggesting that the responsible microbes were probably not strict anaerobes.

Ilag and Curtis (1968) studied ethylene production from 228 species of fungi, and verified ethylene production from about 26% of the species. Aspergillus clavatus produced the greatest amount of ethylene. Research by Lynch (1972) suggested that ethylene was produced by a fungus, Mucor hiemalis, and two yeasts when glucose and methionine were added. Although ethylene is normally produced under anaerobic conditions, the author speculated that when substrates are provided by, for example, crop residues, the production of ethylene may be stimulated by oxygen. Lynch and Harper (1974a, b) further suggested that M. hiemalis, an important colonizer of fresh plant substrates, was a major producer of ethylene in soil and that this organism can grow at relatively low oxygen tensions. They also showed that methionine was a likely precursor of ethylene in soil, where they suggested it might exist as a methionine peptide. Dasilva et al. (1974) showed that ethylene was formed by Mucor and Aspergillus species as well as by an unidentified bacteria and an actinomycete.

Primrose (1976) assayed for ethylene production from bacterial isolates collected from soil and water. Of 756 pure isolates, one-third failed to grow on the defined medium. Most of the isolates (79) producing significant quantities of ethylene were gram-negative, whereas six were gram-positive. Sixty-five of the gram-negative isolates producing ethylene were identified and are shown in Table 4-1, as are the six gram-positive isolates. All isolates produced ethylene only in the presence of methionine, which suggests that it is a precursor for ethylene as was shown for M. hiemalis, Candida vartiovaariai and Trichosporon cutaneum (Lynch 1972) and plants (Yang 1974).

Table 4-1. Identity of ethylene-producing bacteria (from Primrose 1976).

Organism	No. of isolates	Ethylene produced in 66 h	
		mg.(mg dry weight)$^{-1}$	nmol.(mg dry weight)$^{-1}$
Citrobacter sp.	2	1.3-2.3	45-82
Enterobacter cloacae	4	3.1-4.0	111-142
Erwinia herbicola	9	1.1-27	41-950
Escherichia coli	6	2.7-10	97-374
Klebsiella pneumoniae	13	1.2-15	43-546
K. pneumoniae oxytoca	1	9.5	341
K. ozaenae	1	4.1	146
Serratia liquefaciens	1	2.2	78
Aeromonas hydrophila	12	1.1-20	40-722
Pseudomonas group II	4	6.6-15	236-542
Pseudomonas group III or IV	2	17-31	600-1095
P. indigofera	10	18-170	650-6195
Arthrobacter sp.	2	2.3-22	82-800
Saccharolytic coryneforms	4	1.1-11	40-410

NOTE: All isolates are gram-negative except for *Arthrobacter* sp. and saccharolytic coryneforms.

Considine et al. (1977) studied ethylene production from isolates of Penicillium cyclopium and P. crustosum on solid slope media. Penicillium cyclopium produced significantly higher quantities of ethylene when grown on media containing acetate or tricarboxylic acid (TCA) cycle intermediates than when grown on media containing glucose. In contrast, P. crustosum strains showed good ethylene production with glucose. Different regulatory mechanisms and pathways may exist for the two species. Results showed that methionine is not an immediate precursor of ethylene in Penicillium spp. (Considine et al. 1977) as it is in plants and M. hiemalis (Bird and Lynch 1974) and bacteria (Primrose 1976).

Hanke and Dallwet (1976) detected ethylene from 12 of 52 species of fungi from culture, 3 of which were mycorrhizal on the host roots of orchids. The authors suggested that the mycorrhizal fungi may supply ethylene to stimulate the germination of seeds from the host in addition to their other functions. If this is so, these fungi could serve an important ecological role in soil.

Smith and Cook (1974) and Smith (1976) suggested that the main producers of ethylene in soil were spore-forming bacteria in anaerobic microsites in soil. These conclusions were based on the observation that little ethylene was produced from a wide range of soils until most of the O_2 was consumed. Cook and Smith (1977) showed that ethylene production decreased drastically for two soils from saturation to a reduction in soil water potential to -0.5 kPa. These results suggested a greater role for bacteria than for fungi in ethylene production. Further, Smith and Cook (1974) showed that when soil was treated for 30 min with aerated steam at 60, 82, and 100°C to progressively simplify the soil microflora, ethylene production was prevented only when the soil was autoclaved (60 min at 121°C) and it was unaffected when treated at 60 and 82°C. This observation suggested heat activation of spores and hence production of ethylene by spore-forming bacteria. The authors ruled out production by M. hiemalis, since it should be killed by aerated steam at 60°C or above (Baker 1970).

In the debate on the source(s) of ethylene in soil, evidence to support the hypothesis that spore-forming bacteria are the dominant ethylene producers in soil was found by Sutherland and Cook (1980), who showed that ethylene was inhibited by two antibacterial agents (including novobiocin), but not by an antifungal agent (cycloheximide). Since novobiocin, but not cycloheximide, stopped ethylene production in soil heated at 80°C, anaerobic spore-forming organisms and not fungi were suggested to be the dominant producers of soil ethylene. Studies by Lynch (1983), in contrast, showed that treatment of soils or M. hiemalis in pure culture with either novobiocin or cycloheximide had no effect on ethylene production. He suggested that microbial activity as influenced by antimicrobial agents should be viewed with caution, especially for long incubation periods.

In attempting to resolve the debate on sources of ethylene in soil and why ethylene accumulation is usually observed after the depletion of oxygen and the onset of anaerobic conditions, Lynch and Harper (1974a) suggested that the apparent requirement for anaerobic conditions for ethylene accumulation was due to the mobilization of substrates required for ethylene biosynthesis under those conditions, since M. hiemalis grew more rapidly and evolved more ethylene in the presence of O_2. Furthermore, anaerobiosis, which can reduce fungal growth, increases ethylene production (Lynch and Harper 1974b) while decreasing the rate of ethylene degradation (Cornforth 1975).

The evidence now appears to support the hypothesis that fungi are the dominant producers of soil ethylene, especially with regard to studies in pure culture where substantial quantities of ethylene were reported for M. hiemalis (Lynch 1972; Lynch and Harper 1974a), whereas only small quantities were produced by anaerobic bacteria inoculated in autoclaved soil (Sutherland and Cook 1980). However, co-metabolism of precursors by two or more microorganisms cannot be ruled out as a possibility for ethylene production in soil. The range of substrates available is usually much more limiting under cultural than actual soil conditions. Further studies are justified in view of the potential impact of ethylene on other microorganisms and plants.

4.1.1.2 Biochemistry of Biosynthesis

The biosynthesis of ethylene by microorganisms has been studied in various fungi (Bird and Lynch 1974), bacteria (Primrose 1976) and yeasts (Thomas and Spencer 1977). The synthesis of ethylene differs within (Primrose 1976; Chalutz et al. 1977) and among (Considine et al. 1977) microorganisms, and is not the same as in higher plants (Yang 1974). Differences in ethylene biosynthesis are based on its precursors (Lynch 1974; Considine et al. 1977), culture conditions (Chalutz et al. 1977; Mattoo et al. 1979; Kutsuki and Gold 1982), biosynthetic pathways (Wang et al. 1962; Primrose 1976) and their regulatory mechanisms (Chalutz et al. 1978; Thomas and Spencer 1978), and whether ethylene is produced enzymatically (Chalutz et al. 1977), cellularly or extracellularly in the medium (Thomas and Spencer 1977).

Specific precursors to ethylene biosynthesis have been identified as either immediate precursors or compounds that stimulate ethylene production most efficiently in each particular organism. Acetate and TCA cycle intermediates, but not glucose, resulted in more ethylene being produced by the fungus P. cyclopium, whereas P. crustosum produced more ethylene with only glucose (Considine et al. 1977). Penicillium digitatum, the most thoroughly studied fungus in determining ethylene biosynthetic mechanisms, also utilizes acetate efficiently. However, glutamate is an immediate precursor to ethylene in this fungus (Yang 1974). Later studies with P. digitatum showed that methionine, a precursor in higher plants (Baur and Yang 1972), was a precursor as well as an inducer of an ethylene enzymatic system (Chalutz

et al. 1977). Methionine was found to be a precursor of ethylene in the fungus Mucor hiemalis (Lynch 1974), the sulfur-requiring bacteria E. coli strain SPA 0 (Primrose 1976), Pseudomonas fluorescens (Pazout et al. 1981) and the glucose-requiring yeast Saccharomyces cerevisiae (Thomas and Spencer 1977). The fungus Phanerochaete chrysosporium produces ethylene from methanol and 2-keto-4-thiomethylbutyric acid (KTBA) (Kutsuki and Gold 1982), a product derived from ethionine (Thomas and Spencer 1977).

The involvement of glycolysis and the Krebs cycle in ethylene biosynthesis was suggested by Wang et al. (1962) and Ketring et al. (1968) for P. digitatum. Isocitrate was proposed as the branching point from the Krebs cycle where ethylene is derived (Ketring et al. 1968). However, Chou and Yang (1973) later determined that the evidence for isocitrate was a result of a pH effect and that the branching point for ethylene or its precursors was at α-ketoglutarate (KGA) (Yang 1974). Carbons 3 and 4 of glutamate are incorporated into the ethylene skeleton (Wang et al. 1962; Yang 1974); two hydrogens attached to one ethylene carbon are derived from the medium protons and the other methylene group is derived from the methyl group of acetyl-CoA (Chou and Yang 1973).

The regulation of ethylene biosynthesis has been investigated, but remains obscure. Control of ethylene production by inorganic phosphate has been observed in P. digitatum (Chalutz et al. 1978) and P. expansion, in E. coli, and in tissues of higher plants (Chalutz et al. 1980). Pyruvate and glucose have been suggested as regulators of ethylene production in S. cerevisiae (Thomas and Spencer 1977, 1978). Studies conducted with P. digitatum show an inhibition of ethylene production under conditions of high intracellular phosphate (Chalutz et al. 1978). Increased levels of acid phosphatase activity were also observed under low phosphate conditions which were correlated with increased ethylene production (Kapulnik et al. 1983). Greater ethylene production under low phosphate conditions was also accompanied by lower adenosine triphosphate (ATP) levels and higher alkaline phosphatase and protein kinase activities (Mattoo et al. 1979). Regulation of ethylene production did not depend on ATP since levels of adenylates and adenylate energy charge did not mediate phosphate inhibition of ethylene biosynthesis (Mattoo et al. 1983). The regulatory mechanism of ethylene biosynthesis in P. digitatum may involve phosphorylation/dephosphorylation of the ethylene-producing enzyme system mediated through changes in protein kinase activity (Mattoo et al. 1979) and acid phosphatase activity (Kapulnik et al. 1983). This would result in high ethylene production under low phosphate conditions because of depression of the ethylene-synthesizing enzyme system (Mattoo et al. 1979). Regulation of ethylene in S. cerevisiae by glucose may occur in two ways: glucose may reduce the inhibitory effect of pyruvate by decreasing its intracellular concentration and may induce the synthesis of enzymes involved in the biogenesis of ethylene (Thomas and Spencer 1977).

4.1.1.3 Factors Influencing Production

Smith and Dowdell (1974) suggested that temperature, depression of oxygen, availability of substrates and the effect of soil moisture on air-filled porosity of soil (and thus on the rate with which oxygen diffuses into and ethylene out of soil pores) all affect the production of ethylene. In agricultural soils, residue management, tillage, and fertilization practices may also affect ethylene accumulation by the manipulation of available substrates, soil nutrients and soil physical factors.

4.1.1.3.1 Temperature and Sterilization

A progressive increase in ethylene production by soil was observed between 5 and 35°C by Smith and Restall (1971). Smith and Dowdell (1974) later showed a significant relationship between soil temperature and ethylene in the range of 4 to 11°C. The concentration of ethylene increased 20 times in this range; this was attributed to an increase in the fraction of the total soil volume that is anaerobic, i.e. the centers of aggregates became progressively more anaerobic with the increase in temperature, and the subsequent consumption of oxygen increased at the periphery of aggregates.

Sutherland and Cook (1980) showed that aerated steam at 80°C for 30 min failed to reduce ethylene production by soil incubated at 30°C. Autoclaving the soil twice at 121°C (60 min, then 20 min) prevented ethylene production. These results were used to suggest that the major source of ethylene in soil was spore-forming bacteria, which would not be killed by aerated steam at 80°C. However, nonenzymatic formation of ethylene from heated organic matter is also a possibility (Rovira and Vendrell 1972). Lynch (1975), who showed that ethylene was produced from heat-killed cultures of M. hiemalis, speculated that the organism produced a heat-labile intermediate which nonenzymatically produced ethylene.

The effect of soil drying was studied by Goodlass and Smith (1978a) who showed that air drying and, to a greater extent, oven drying at 105°C enhanced ethylene production. This production was correlated with organic matter content. However, the increase in "evolution" of ethylene following air and oven drying may reflect the reduction in ethylene decomposition upon drying, as was shown by Cornforth (1975).

Rovira and Vendrell (1972) showed that ethylene was produced in relatively large amounts from soil sterilized by gamma radiation. Although concentrations of other hydrocarbons also increased, only ethylene levels increased to levels potentially toxic to plants.

4.1.1.3.2 Amendments and Pesticides

Since the general microbial activity of the soil is positively influenced by its level of organic matter, it is reasonable to assume that soils higher in organic matter will produce more ethylene. Goodlass and Smith (1978b) showed that the addition of barley and wheat straw, but not hay, to anaerobic soil resulted in increased ethylene concentration. Total soil organic matter, and not previous history, was also suggested as important in ethylene production (Goodlass and Smith 1978a). The production of ethylene, although higher for topsoil than for subsoil, was proportional to the quantity of organic matter present. No relation was obtained between grassland and arable soils.

Phenolic acids (degradation products of soil organic matter) were shown to promote ethylene production by a soil isolate, P. cyclopium (Considine and Patching 1975). Organic acids also promoted ethylene production by strains of both P. cyclopium and P. crustosum in cultures, suggesting a possible reason for increased ethylene from higher levels of soil organic matter where degradable levels of phenolic polymers are found. Ethylene production was also enhanced where animal wastes were applied to soil (Burford 1975).

Insecticides (Carbofuran and Endosulfan) had no effect on the production of ethylene whereas the application of two fungicides (Captan and Thiram) led to increased concentrations of ethylene in the soil atmosphere in laboratory studies (Wainwright and Kowalenko 1977). This was perhaps due to an increase in available substrate for ethylene producers after the lysis of cells.

The addition of lime increased ethylene production, as did chitin and cellulose (Wainwright and Kowalenko 1977). A possible role of actinomycetes in the production of ethylene was suggested, since chitin often promotes the growth of actinomycetes.

4.1.1.3.3 Oxygen and Waterlogging

Smith and Dowdell (1974) showed that there was a relationship between high moisture content and both the production of ethylene and the depression of oxygen. Smith and Russell (1969) showed that the presence of O_2 was directly related to the accumulation of ethylene in an open or closed atmospheric environment.

Lynch (1972) showed that the decline in ethylene production after waterlogging during winter can be overcome by the addition of glucose and methionine. This suggests that the production of ethylene is limited by the availability of substrates. Hunt et al. (1980) reported a positive and significant correlation of ethylene production with organic matter in coastal plain soil from the southeastern United States.

In an attempt to relate chlorosis of wheat to winter waterlogging in southwestern Australia, Primrose and Dilworth (1976) isolated several strains of bacteria from soil (pseudomonads, Enterobacter aerogenes and Escherichia coli) and calculated the concentration of ethylene in the soil water atmosphere. It was not shown if the enteric organisms that produced ethylene in culture under aerobic conditions could produce ethylene under anaerobic conditions in the field.

Ethylene production increased in soil as oxygen levels decreased in relation to the level of soil flooding (Hunt et al. 1981). The air in the soil generally contained less than 1 $mg.m^{-3}$ ethylene when it contained more than 9% O_2. However, when the air in the soil contained less than 9% O_2 and when the Eh was greater than +150 mV, only 4% of the samples had less than 1 $mg.m^{-3}$ ethylene, and less than 5% of the samples contained more than 7 $mg.m^{-3}$ ethylene. At less than 9% O_2 and at 150 mV Eh or less, 16% of the samples contained more than 7 $mg.m^{-3}$ ethylene. Under less reduced conditions and between 5 and 10% O_2, significant quantities of ethylene can accumulate in coastal plain soils in the southern United States.

Hunt et al. (1982) reported more ethylene accumulation in soil held at saturation than at 5 kPa metric water potential. Total ethylene accumulation ranged from 0.17 to 5.33 $mg.L^{-1}$ under saturation for 21 soils. Half of the samples produced more than 1 $mg.L^{-1}$ ethylene which is a level reported to damage sensitive plants (Jackson and Campbell 1975, 1976b; Jackson et al. 1978).

4.1.1.3.4 Soil Redox Potential, pH and Nitrate

Hunt et al. (1982) showed that ethylene accumulated to a greater extent in soil under reduced (Eh = 0 mV) conditions than under oxidized (Eh > 250 mV) conditions. That may reflect the greater tendency for ethylene to degrade under the more oxidized conditions (Cornforth 1975). Goodlass and Smith (1978a) showed that for 19 fresh soils there was no relation between soil pH and the production of ethylene. However, a fivefold increase in ethylene production, when expressed per unit of organic carbon of air-dried soils, was observed with a pH decrease from neutrality to pH 5.0. In general, ethylene production is expected to be optimum at or near neutrality, since soil microbial activity is usually optimum in the neutral pH range.

Hunt et al. (1980) showed that additions of up to 100 mg of NO_3^--N per kilogram of soil reduced ethylene production briefly when Bermuda grass residues were incorporated in soil with nitrate. Smith and Cook (1974) suggested that there was a negative relation between soil nitrate level and ethylene production, since high levels of nitrate in agricultural soils would cause the redox potential to reach a value above which ethylene production begins. That was one reason why

ethylene production was suggested to be lower for cultivated than for noncultivated soils. In contrast, no relation was obtained between ethylene production and the nitrate level in fresh soil, especially when the variable organic matter was eliminated from the regression (Goodlass and Smith 1978a). The production of ethylene was inhibited by nitrate addition to air-dried soil. The authors concluded that the nitrate level in the soil would be of little consequence in delaying the production of ethylene.

4.1.2 PLANTS

4.1.2.1 Production Rates

Ethylene is produced by most, if not all, plants and parts of plants. The rate of normal ethylene production differs with the kind of plant, with the part of the plant and with the stage of development. For example, quantities of ethylene evolved by ripening fruits range from 0.05 $\mu L.kg^{-1}.h^{-1}$ (1 μL = 40.8 nmol) for Valencia oranges to 350 $\mu L.kg^{-1}.h^{-1}$ for passion fruit. Faded Vanda orchid blossoms are among the highest producers known, evolving 3400 $\mu L.kg^{-1}.h^{-1}$. For most biological tissues, ethylene production has been reported to be from 0.5 to 6 $\mu L.kg^{-1}.h^{-1}$ (Leopold 1972). The appropriate time for harvesting of fruit can be accurately assessed by determining the ethylene production in the orchard (Dilley 1982).

Details on the production of ethylene by plants are available in a number of reviews (e.g. Porritt 1951; Spencer 1969; Mapson 1969; Abeles 1973; Yang and Hoffman 1984). Data on ethylene production by trees, bushes and aquatic plants are limited.

A meaningful estimate of biological production of ethylene in Canada is not possible at present because of the lack of such information as the amounts of ethylene evolved in different seasons and climatic zones by forests of various kinds, by various agricultural crops, by arctic and alpine tundra, by meadows and by improved and unimproved rangeland and other vegetated land. In some cases, even figures on the amounts of vegetation are difficult to obtain. With the large areas of water within Canada, the production of ethylene by aquatic organisms should not be ignored. An ethylene balance should also consider production and consumption by soil microorganisms under different conditions, but extensive knowledge in this area is lacking everywhere in the world. Abeles *et al.* (1971) has estimated that plants in the United States produce 2×10^{4} $t.a^{-1}$ of ethylene. (This was calculated on assumptions of 0.5 μL (20 nmol) of ethylene produced per kilogram (fresh weight) every hour of the year and of 2.2 t of vegetation per hectare and an area of 7.8×10^{6} km^2 for the United States.)

Most measurements of the rate of ethylene production by plants reported in the literature are based on techniques that are subject to

problems (see Chapter 3). These problems include elevated "wound ethylene" production by excised tissues, and limitations associated with the use of closed systems. As an example of the former, Bassi and Spencer (1983a, b) showed that the supposed effect of light on ethylene production was an artifact resulting from the use of leaf discs. Light does not affect ethylene production in whole plants. There are at present few reliable figures on the production of ethylene by plants, even under laboratory conditions.

In addition, the rate of ethylene production in plants can increase severalfold in response to various environmental stresses (see also Table 7-6, Section 7.2.4.2). Those stresses are summarized by Abeles (1982) and include disease (e.g. Pegg 1976; Boller 1982), wounding, the application of many herbicides or auxin (e.g. Morgan 1976), exposure to a variety of air pollutants (e.g. Tingey et al. 1976; Peiser and Yang 1978; Bressan et al. 1979), attack by insects, drought (e.g. McMichael et al. 1972), flooding (e.g. Kawase 1974) and exposure to temperature extremes or mechanical stress such as from wind or obstructions (e.g. Jaffee 1970). Therefore, the commonly cited data for the rates of ethylene production may not represent an accurate assessment of the ethylene produced by the plants under natural conditions.

4.1.2.2 Biosynthetic Pathways

The biosynthetic pathway for ethylene in ripening apples (Adams and Yang 1979), soybean leaves (Lurssen et al. 1979) and a number of other higher plant tissues (Cameron et al. 1979; Yang et al. 1980) is as follows: S-adenosylmethionine (SAM) is split to produce 5'-methylthioadenosine (MTA) and 1-aminocyclopropane-1-carboxylic acid (ACC). The latter is then converted to ethylene as shown in Figure 4-1. Adenosine is rapidly hydrolyzed from MTA, producing 5-methylthioribose (MTR), which then forms methionine (Yang et al. 1982). Thus, the CH_3-S- group of methionine is conserved for continued synthesis of ethylene, whereas the 4-carbon moiety of methionine, from which the ethylene molecule is derived, is ultimately replenished from the ribose moiety of ATP. This cycle is of particular importance in those tissues that produce ethylene at high rates but contain very low concentrations of endogenous methionine (Yang 1983). Characteristics of the enzyme system converting ACC to ethylene and other details are described by Yang et al. (1982). Boller et al. (1979) and Yu et al. (1979) have prepared a soluble enzyme (from tomato fruit) capable of converting SAM to ACC. The enzyme that catalyzes the conversion of ACC to ethylene has not been isolated in a cell-free form. Free radicals appear to be involved as intermediate reactants (Apelbaum et al. 1981).

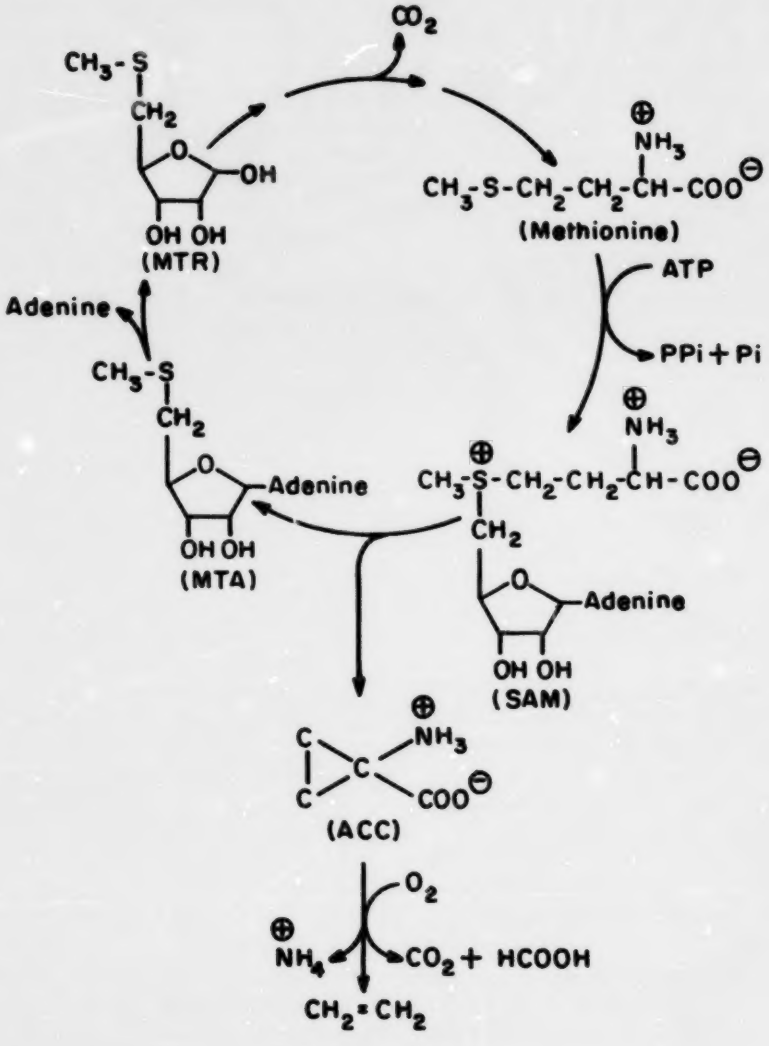


Figure 4-1. Methionine cycle in relation to ethylene biosynthesis in apple fruit (from Yung et al. 1982). Abbreviations: ACC, 1-aminocyclopropane-1-carboxylic acid; MTA, 5'-methylthioadenosine; SAM, S-adenosylmethionine; MTR, 5-methylthioribose.

Increased ethylene synthesis is a result of enhancement of the activity of ACC synthase when a tissue is wounded (Boller and Kende 1980; Yu and Yang 1980) or when it is treated with the naturally occurring growth substance, indole-3-acetic acid (Jones and Kende 1979; Yu and Yang 1979). In mung bean hypocotyls, detectable amounts of ACC are formed only when the tissue is treated with indole-3-acetic acid at concentrations that induce ethylene production (Imaseki et al. 1982). From the same tissues, a protein that reversibly inhibits auxin-induced ethylene production was isolated. The inhibited step is the conversion of ACC to ethylene. Similar proteins have been found in seeds of several legumes and in marine algae.

In tissues such as wilted leaves or germinating seeds (but not, for example, in postclimacteric apples), ACC can be converted, apparently irreversibly, to 1-(malonylamino)-cyclopropane-1-carboxylic acid (Amrhein et al. 1982; Hoffman et al. 1982). Yang et al. (1982) view this conversion as a detoxification process, reducing the amounts of ethylene formed.

Recent work, summarized by Galston (1983), shows that ethylene and polyamines arise from a common precursor, SAM, and suggests that their formation can be competitive under certain situations. (Polyamines have been proposed to be second messengers for the action of a number of plant growth hormones.) There appears to be a system of feedback controls, since polyamines inhibit ethylene production and ethylene inhibits polyamine production.

Some evidence exists for other pathways for the biosynthesis of ethylene by higher plants (e.g. Stinson and Spencer 1969; Shimokawa and Kasai 1970a, b; Ghooprasert and Spencer 1975). There is evidence that the proportions of ethylene produced by different pathways vary at different stages of an organism's development or under different environmental conditions (Knight 1970).

4.1.3 HUMANS AND OTHER ANIMALS

Ethylene is also produced by animals, though relatively little research has been done in this area. Chandra and Spencer (1963a) showed that the ethylene content of the exhaled gas of fasted, adult nonsmokers was nearly double that of the inhaled air (~4.0 $\mu g.m^{-3}$; 3.5 ppb). Exhalation of ethylene by humans has been confirmed (Conkle et al. 1975; Harrison 1981). Sagai and Ichinose (1980) found that the measurement of ethylene exhaled by rats, as well as that of the hydrocarbons ethane, pentane and butane, was a sensitive index of *in vivo* lipid peroxidation during aging, the amount exhaled being related to age with a linear regression fit correlation coefficient. Ando et al. (1983) also found that ethylene and ethane were expired by mice and rats, and suggested the amounts exhaled were representative of the extent of *in vivo* lipid peroxidation. It has been suggested that

pollutants other than ethylene may contribute to the *in vivo* production of ethylene, e.g. via the initiation of lipid peroxidation by NO_2 (Törnqvist 1984). Kakanov (1960) detected ethylene in air passed through respiratory chambers that contained rats or swine ascarids, but no precautions were taken to maintain aseptic conditions.

The production of ethylene observed in the experiments described above was probably not solely attributable to the gut flora, since subcellular fractions from rat liver and rat intestinal mucosa were shown to produce ethylene (Chandra and Spencer 1963b). Fu *et al.* (1979) found evidence of the enzymatic formation of ethylene from methionine and α-keto-4-thiomethylbutyric acid by rat liver extracts, suggesting that those compounds can serve as precursors in this system. The conversion of methionine to ethylene was dependent on the presence of oxygen, which could indicate that oxidase-like reactions are involved. The authors suggested that the production of ethylene could be involved physiologically in animal metabolism, because of its increase with growth of the animal.

Kakanov (1960) reported that the formation of subcutaneous abscesses, or subcutaneous transplantation of tumors, resulted in an increased ethylene content of the gases exhaled by rats. Chandra and Spencer (1961) found that cultures of tumor tissue produced much larger amounts of ethylene than did those of normal tissue. Other workers (Gibson 1963; Lieberman and Hochstein 1966) have also shown the production of ethylene by subcellular fractions from rat liver.

There has been no further work on the pathways for ethylene biosynthesis in animals, or on the regulation of its production.

4.2 ANTHROPOGENIC

In Canada, information is fragmentary concerning the quantities of ethylene emitted from anthropogenic sources. The emission estimates presented here rely principally on estimates of emissions of total volatile organic compounds (VOC). This information, coupled with available data on the ethylene fraction of VOC emissions extracted from published information, forms the basis of the emission calculations. Consequently they are to be treated as preliminary estimates which are subject to further refinement.

Anthropogenic emissions of ethylene originate mainly from two sources: fuel combustion and industrial petrochemical activity. Other sources do not appear to contribute significantly to the ethylene emissions in Canada.

N.B. THE EMISSION ESTIMATES PRESENTED IN THE SECTIONS THAT FOLLOW ARE FOR THE BASE YEAR 1980.

4.2.1 EMISSIONS FROM FUEL COMBUSTION

Fuel combustion emissions can be separated into three distinct areas: stationary combustion sources, waste incineration, and transportation. The sources that are considered in each of these categories are as follows:

(i)	Stationary combustion sources:	Thermal power stations
		Industrial fuel combustion
		Commercial fuel combustion
		Residential fuel combustion (excluding wood)
		Fuelwood combustion
(ii)	Waste incineration:	Agricultural waste burning
		Municipal incinerators
		Industrial and commercial incinerators
		Wigwam burners
		Slash burning
(iii)	Transportation:	Gasoline-powered motor vehicles
		Off-road use of gasoline
		Diesel-powered engines
		Railroad sector
		Marine sector
		Aircraft

4.2.1.1 Stationary Combustion Sources

The combustion of fossil fuels for such purposes as electric power generation, space heating, and the production of steam in industry produces emissions of VOC. The major fuels consumed are coal, heavy oil, light oil, diesel oil and natural gas (fuelwood combustion is treated separately below). Emissions of ethylene have only been reported from the combustion of diesel oil in small generators and turbines. Ethylene accounts for about 29% by weight of the VOC emissions from this source (GCA 1980). Based on this percentage and the estimated 920 Mg (1 Mg = 1 tonne) of VOC released from diesel-powered generation sources (Environment Canada 1983), the emissions of ethylene amounted to 170 Mg. Ethylene does not appear to be released from the combustion of other fuels in stationary sources (GCA 1980).

Various forms of wood combustion processes are addressed in this report, including slash fires and wigwam burners (Section 4.2.1.2), and fuelwood used in fireplaces and various residential heating stoves (below). Similarities in the types of organic compounds evolved might be expected, and there is some evidence to support such similarities (Eccleston *et al.* 1974; Ward *et al.* 1976; Butcher and Sorenson 1979; Hall and DeAngelis 1980). Therefore, in view of the limited data available, ethylene emissions from the various forms of wood combustion have been based on results from slash burning. Data provided by

Fritschen et al. (1970) and Sandberg et al. (1975) show that ethylene accounts for about 7.5% by weight of the VOC emissions from this form of wood combustion. It should be noted, however, that the various wood combustion processes release VOC at different rates. Table 4-2 summarizes the emission factors for wood combustion processes.

Based on the above and the amount of fuelwood consumed in fireplaces and residential heating stoves (Statistics Canada 1980a), ethylene emissions are estimated to be 4800 Mg.

4.2.1.2 Waste Incineration

Burning of vegetation, including agricultural waste, produces ethylene (see Section 4.2.4 for a discussion of ethylene emissions from tobacco burning). The amount of ethylene produced appears to be a function of moisture content, type of vegetation, and location (Darley et al. 1966). Boubel et al. (1968) determined ethylene emission rates from burning various grass stubble to be in the order of 0.3 to 1.5 $kg.Mg^{-1}$. Other vegetation-based waste material yielded between 0.1 and 2.7 $kg.Mg^{-1}$ (Darley et al. 1966). Abeles (1982) estimated a conversion factor for ethylene production from vegetation fires to be about 1 $kg.Mg^{-1}$.

Burning of agricultural waste is mostly a seasonal practice in Canada. Postharvest stubble field burning is often practised and is unquestionably a source of ethylene emissions; however, no information on the quantity of agricultural waste burned in Canada is available. Gerakis et al. (1978) reported air ethylene concentrations between 2 and 33 $mg.m^{-3}$ 1 m above burning wheat stubble. They also measured some elevation of ambient air ethylene levels further from the fire. McElroy (1960) recorded 5 $mg.m^{-3}$ ethylene in smoke of rice stubble burns, with levels dropping to 350-575 $\mu g.m^{-3}$ 30 m above the burn. During a barley stubble burn, levels were 115-460 $\mu g.m^{-3}$ at a distance of 2-8 km, not much higher than the pre-burn concentration. No data are available for ambient air concentrations of ethylene resulting from agricultural waste burning in Canada.

Ethylene has been detected in the pyrolysis products of solid municipal waste (Jerman and Carpenter 1968). Concentrations of up to 11.5 $mg.m^{-3}$ have been reported in multiple-chamber incinerator effluents (Tuttle and Feldstein 1960). Single-chamber incinerator effluent contains between 23 and 34 $mg.m^{-3}$ of ethylene (Feldstein et al. 1959).

From an intercomparison of several sets of stack effluent measurements of multiple-chamber incinerators (Tuttle and Feldstein 1960; Feldstein et al. 1963; Hangebrauck et al. 1964; Carotti and Kaiser 1972; Carotti and Smith 1974; McColgan 1977), an average value of 11% by weight ethylene in VOC emissions from municipal incinerators was found. This percentage, combined with the VOC emission rate from municipal

Table 4-2. Estimated emission factors for wood combustion.

Wood combustion process	Emission factors ($kg.t^{-1}$)	
	Volatile organic compounds[α] (VOC)	Ethylene[β]
Fuelwood combustion (residential heating)	15.0	1.13
Slash burning	2.0	0.15
Wigwam burners	5.5	0.41

[α] U.S. EPA (1977).

[β] Derived from the assumption that 7.5% by weight of VOC emissions are in the form of ethylene.

incinerators (U.S. EPA 1977), yields an ethylene emission factor of 0.08 $kg.t^{-1}$ of refuse burned.

Based on the above and the quantity of refuse burned in Canada (Environment Canada 1984), ethylene emissions from municipal incinerators are estimated to be 70 Mg. Ethylene emissions from industrial and commercial incinerators are negligible.

The forestry industry produces ethylene emissions when wood waste material is incinerated in wigwam burners, as well as during slash burning operations. The amount of wood waste burned in wigwam burners was calculated based on the production of softwood and hardwood lumber (Environment Canada 1975; Statistics Canada 1980b). Based on the values shown in Table 4-2 and the amount of wood waste burned in wigwam burners, ethylene emissions are estimated to be 2300 Mg.

Slash is burned to reduce the hazard of forest fires, control insects, and provide favorable growing conditions for seedlings. The quantity of slash burned in Canada was calculated based on primary forest production (Statistics Canada 1980b) and a conversion factor of 1 t of slash per 63 050 m^3 of forest production. Using the emission factor derived in Table 4-2 and the estimate of the quantity of slash burned, ethylene emissions are estimated to be 1500 Mg.

4.2.1.3 Transportation

Gasoline-powered motor vehicles are by far the largest source of ethylene emissions in Canada. They include all vehicles driven on public roads using gasoline as fuel and can be classified in four major categories: automobiles, light-duty trucks, medium-duty trucks and heavy-duty trucks.

Estimates of emissions of VOC were based upon vehicle registration figures, the age distribution of Canadian vehicles, estimated speed distribution, emission rate degradation factors and estimated vehicle kilometres traveled per year (Gourly 1984). The emission factors in terms of grams per kilometre were converted to kilograms per vehicle per year on the assumption that automobiles travel 17 220 $km.a^{-1}$, light-duty trucks 21 420 $km.a^{-1}$, medium-duty trucks 22 530 $km.a^{-1}$ and heavy-duty trucks 27 040 $km.a^{-1}$. Vehicle registrations were obtained from Statistics Canada (1980c).

Results obtained by the U.S. EPA (1980a) from emission tests show that ethylene emissions constitute 5.6 and 11.6% of the VOC emissions from automobiles with and without catalytic converters, respectively. Table 4-3 presents a summary of the emission factors for gasoline-powered motor vehicles.

Based on the emission factors shown in Table 4-3 and vehicle registration figures, the emissions of ethylene are estimated to be

Table 4-3. Estimated emission factors for gasoline-powered motor vehicles.

Vehicle type	Emission factors ($kg.a^{-1}$ per vehicle)	
	Volatile organic compounds[α] (VOC)	Ethylene
Automobiles	43.1	3.8[β]
Light-duty trucks	49.4	4.4[β]
Medium-duty trucks	79.9	9.3[γ]
Heavy-duty trucks	265.8	30.8[γ]

α Estimated by Gourly (1984).

β Derived from the assumption that 45% of the in-use automobiles and light-duty trucks are equipped with catalytic converters (Gourly 1984).

γ Based on an ethylene-to-VOC ratio of 11.6% for noncatalyst-equipped automobiles.

39 000 Mg for automobiles, 6600 Mg for light-duty trucks, 8000 Mg for medium-duty trucks and 9600 Mg for heavy-duty trucks. In total, ethylene emissions from gasoline-powered motor vehicles are estimated to be 63 200 Mg.

Approximately 12% of the gasoline sold in Canada was not consumed on public roads and highways (Statistics Canada 1980d), but was used mainly in construction, agricultural and forestry equipment. The emissions of VOC from these sources were estimated based on the amount of fuel consumed and emission factors developed by the U.S. EPA (1977). Data specific to ethylene were not available; however, ethylene emissions can be approximated by using the ethylene-to-VOC emissions ratio for noncatalyst-equipped automobiles (Table 4-3). Table 4-4 presents a summary of the emission factors for the off-road use of gasoline.

Based on the emission factors derived above and the amount of fuel consumed in off-road use of gasoline, ethylene emissions are estimated to be 5600 Mg.

Diesel fuel is consumed by heavy-duty diesel trucks, railway locomotives, steamships, and construction and agricultural equipment. Data on the emissions of ethylene from transportation sources burning diesel are limited. An estimate of the chemical composition of VOC emissions from diesel combustion has nevertheless been made. The ratio of ethylene to total VOC emissions has been reported as 10.7% by weight (U.S. EPA 1980a). Table 4-5 presents a summary of the emission factors estimated for diesel combustion in transportation. They are generally given as a function of fuel consumption.

For steamships, the number of calls in port was estimated from the number of ships at Canadian ports for coastwise and international shipping (Statistics Canada 1980e). The average time that a ship remains in port (per call) is 2.66 d (Gaudreau 1984). The consumption of diesel fuel for the various sources mentioned above was obtained from Statistics Canada (1980c, f).

Based on the emission factors calculated above and the amount of diesel fuel consumed, the emissions from diesel consumption in transportation are estimated to be 15 500 Mg.

Information was not available on emissions of ethylene from aircraft engines. If it is assumed that the same ratio of ethylene to total VOC emissions ratio that was used for noncatalyst-equipped automobiles (11.6%) applies, the resulting ethylene emissions would be 1100 t. This is based on VOC emissions from air transport of 9100 t (Environment Canada 1983).

Table 4-4. Estimated emission factors for off-road use of gasoline.

Source	Emission factors ($kg.10^{-3}$ L fuel)	
	Volatile organic compounds[α] (VOC)	Ethylene[β]
Agricultural equipment	16.8	1.9
Heavy-duty construction equipment	28.1	3.3
Industrial engines (mainly forestry equipment)	27.4	3.2

[α] U.S. EPA (1977).

[β] Derived from the assumption that 11.6% by weight of VOC emissions are in the form of ethylene.

Table 4-5. Estimated emission factors for diesel-powered engines.

Source	Emission factors	
	Volatile organic compounds[α] (VOC)	Ethylene[β]
Railway locomotives	11.0 $kg.10^{-3}$ L	1.2 $kg.10^{-3}$ L
Steamships		
Dockside	39.8 kg per call in port	4.3 kg per call in port
At sea	12.0 $g.L^{-1}$	1.3 $g.L^{-1}$
Heavy-duty trucks	157.0 $kg.a^{-1}$ per vehicle	16.8 $kg.a^{-1}$ per vehicle
Agricultural vehicles	7.3 $kg.10^{-3}$ L	0.8 $kg.10^{-3}$ L
Construction vehicles	3.6 $kg.10^{-3}$ L	0.4 $kg.10^{-3}$ L
Other diesel engines	4.5 $kg.10^{-3}$ L	0.5 $kg.10^{-3}$ L

[α] U.S. EPA (1977).

[β] Estimated on the assumption that 10.7% by weight of VOC emissions are in the form of ethylene.

4.2.2 EMISSIONS FROM INDUSTRIAL PROCESSES

In Canada, ethylene is manufactured through thermal cracking of naphtha or ethane feedstocks. Refinery gas also contains about 5-10% ethylene and sometimes serves as a source.

Canadian facilities for the production of ethylene, in three locations, had a combined total nameplate capacity for 1982 of 1 540 000 $Mg.a^{-1}$, up from 1 200 000 Mg in 1980 (Corpus 1982). The plants are in Sarnia, Ontario, with a total nameplate capacity of 700 000 $Mg.a^{-1}$; Montreal, Quebec, with an annual 300 000 Mg capacity; and Joffre, Alberta, with a capacity of 540 000 $Mg.a^{-1}$ (Corpus 1982). The Joffre plant is scheduled to be expanded by 680 000 $Mg.a^{-1}$ in 1984. Ethylene from these facilities is either used on the same site or shipped by pipeline to derivative plants located within Canada. Storage is in underground caverns using brine displacement (Corpus 1982).

Ethylene is a major growth petrochemical in Canada, being the raw material for a diverse petrochemical derivatives industry (Figure 4-2). Plastics, such as polyethylene and poly(vinyl chloride), are major end products. Other important chemicals produced from ethylene are ethylene oxide, ethylene glycol, ethanol and styrene (through ethylbenzene).

Under normal operating conditions, petrochemical plants using or producing ethylene do not have distinct ethylene emission points. Unusual process conditions or upsets may result in the release of ethylene to the atmosphere through emergency venting or incomplete combustion of flared gases. Another important source of ethylene (and other VOCs) from industry is through fugitive emissions - those that originate from numerous minor sources such as flange, valve and pump seal leaks. Emissions can also occur during the storage of ethylene.

An analysis of ethylene emissions from Canadian production plants was conducted by the Chemical Industries Division of Environment Canada (Madé 1984). The emissions estimate was based on ethylene production values (Corpus 1982) and total VOC emission rates for the different production prccesses as developed by the U.S. EPA (1980b). A ratio of 15% ethylene to total VOC emissions was used to calculate the ethylene component of the VOC emissions. This value was determined by an analysis of the emissions components of about 40 production processes in the petrochemical industry. On average, it was found that 15% of the VOC emissions were made up of the petrochemical being produced. Based on this analysis, emissions are estimated to be 1800 Mg from the production of ethylene.

Emissions from the production of ethylene-derived petrochemicals were estimated using a methodology similar to the above.

In this case, the ethylene-to-VOC emission ratios were known for the various production processes (U.S. EPA 1980b). Ethylene emissions from secondary processing amounted to 23 200 Mg in 1980. Information on emission factors is presented in Tables 4-6 and 4-7 for ethylene production and ethylene-derived petrochemicals.

It should be noted that the emissions estimates developed for the production of ethylene and ethylene-derived petrochemicals do not account for any control procedures that may be in place in the industry, e.g. the practice of incinerating emergency gas releases through flares, which is now standard. This practice would in effect lower the emissions of the ethylene from this industry and consequently the emission estimates reflect only potential emissions or uncontrolled emissions. Actual emissions could be significantly lower.

Other industrial sources of ethylene emissions in Canada are gasoline marketing, coke manufacturing and petroleum refining.

Emissions from gasoline marketing facilities involve losses caused by product transfer activities (loading-unloading) and the losses associated with storage tanks at terminal sites. Using VOC emission factors from these operations as developed by the U.S. EPA (1977), gross sales of gasoline (Statistics Canada 1980d) and an ethylene-to-VOC ratio of 0.03% (Trijones and Arledge 1976), ethylene emissions from gasoline marketing are estimated to be 70 Mg.

Coke is formed in ovens by driving off the volatile constituents of coal such as tar, benzene and naphthalene. In Canada, coke is manufactured primarily by the by-product process which involves heating coal in rectangular coking ovens grouped together in a series and alternately interspersed with heating flues. Emissions of VOC and ethylene were calculated using information on the quantities of coke produced (Aylsworth 1981), VOC emission factors (Choquette 1974), and an ethylene-to-VOC ratio of 28% (U.S. EPA 1980a; Bounicore 1980; Allen 1980). Based on the above, ethylene emissions are estimated to be 500 Mg.

The composition of emission streams from oil refineries differs widely from refinery to refinery. Generally, the major sources of ethylene are the catalytic cracking units, the carbon monoxide boilers and the coking units (U.S. EPA 1980a). Based on the above, and information developed by Environment Canada on VOC emissions from petroleum refineries (Environment Canada 1984), ethylene emissions are estimated to account for less than 1% of total VOC emissions, or about 1300 Mg.

4.2.3 EMISSIONS FROM AGRICULTURAL AND FORESTRY APPLICATIONS

Ethylene is often used to ripen fruit in storage chambers. Fruits like tomatoes and bananas are commonly shipped green to Canada.

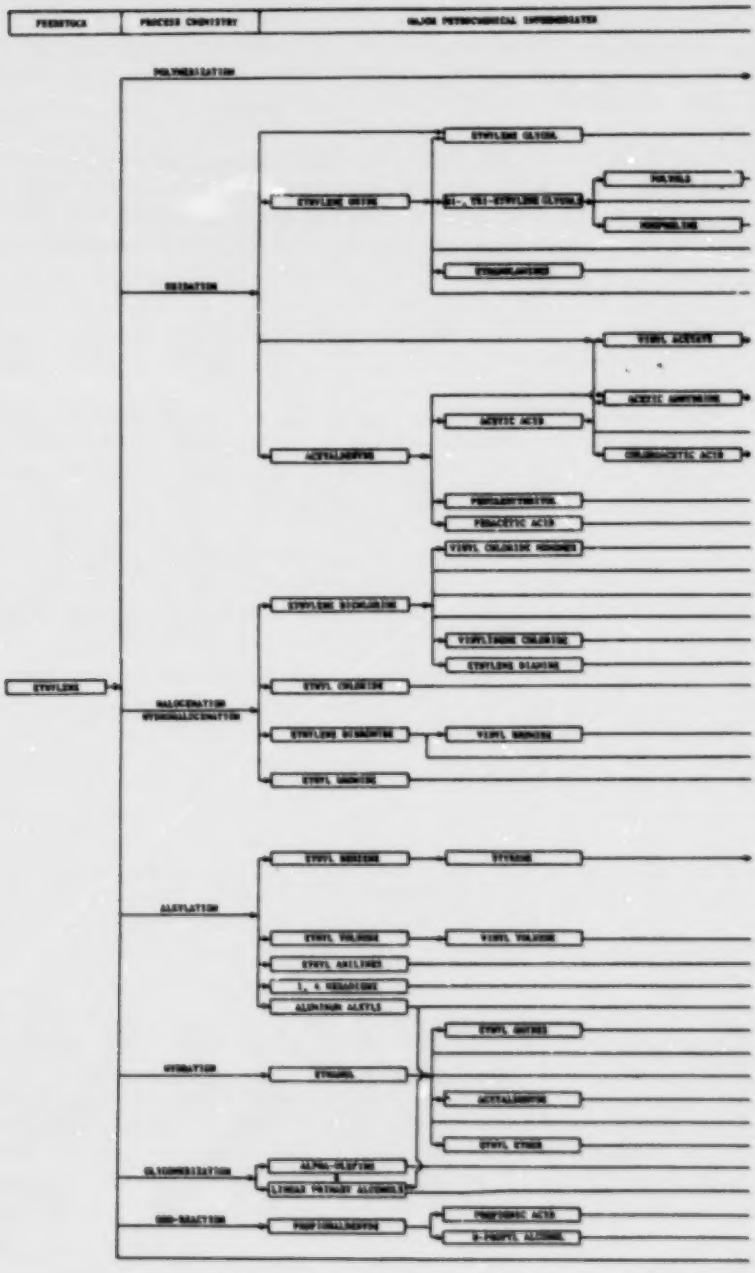
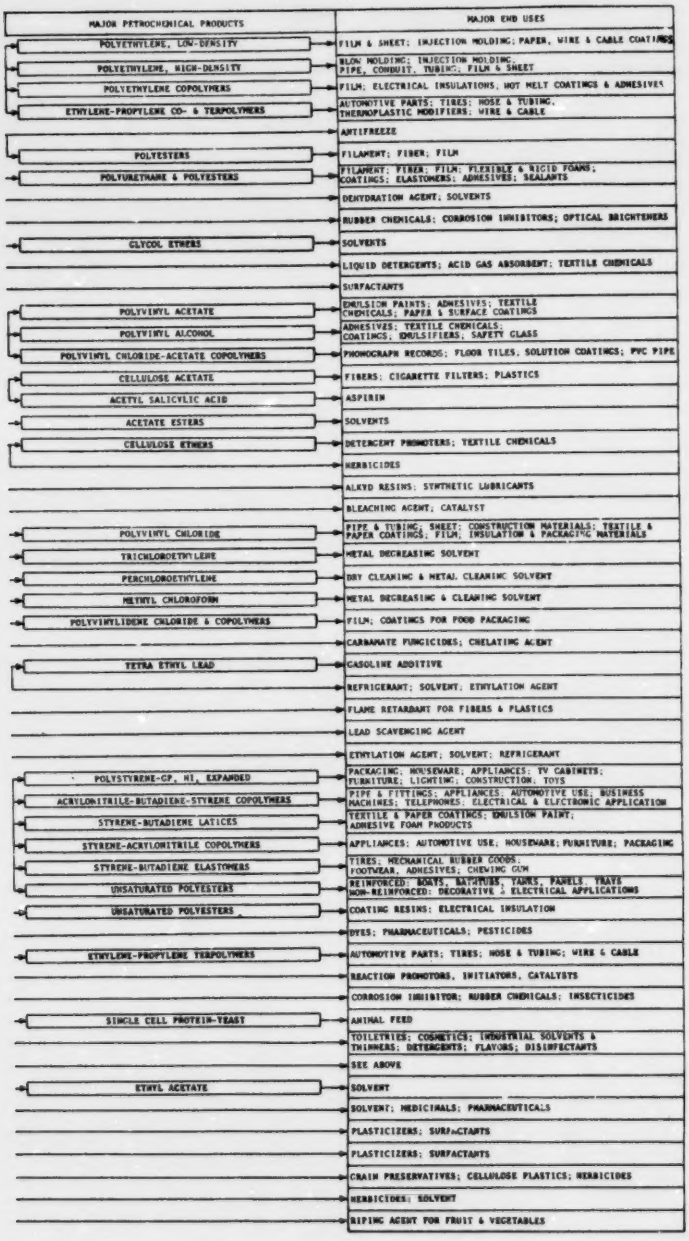


Figure 4-2. Major ethylene intermediates, petrochemical products and Marcel Dekker, Inc.).


MAJOR PETROCHEMICAL PRODUCTS
MAJOR END USES
POLYETHYLENE, LOW-DENSITY
FILM & SHEET; INJECTION MOLDING; PAPER, WIRE & CABLE COATINGS
POLYETHYLENE, HIGH-DENSITY
BLOW MOLDING; INJECTION MOLDING; PIPE, CONDUIT, TUBING; FILM & SHEET
POLYETHYLENE COPOLYMERS
FILM; ELECTRICAL INSULATIONS; HOT MELT COATINGS & ADHESIVES
ETHYLENE-PROPYLENE CO- & TERPOLYMERS
AUTOMOTIVE PARTS; TIRES; HOSE & TUBING; THERMOPLASTIC MODIFIERS; WIRE & CABLE
ANTIFREEZE
POLYESTERS
FILAMENT; FIBER; FILM
POLYURETHANE & POLYESTERS
FILAMENT; FIBER; FILM; FLEXIBLE & RIGID FOAMS; COATINGS; ELASTOMERS; ADHESIVES; SEALANTS
DEHYDRATION AGENT; SOLVENTS
RUBBER CHEMICALS; CORROSION INHIBITORS; OPTICAL BRIGHTENERS
GLYCOL ETHERS
SOLVENTS
LIQUID DETERGENTS; ACID GAS ABSORBENT; TEXTILE CHEMICALS
SURFACTANTS
POLYVINYL ACETATE
EMULSION PAINTS; ADHESIVES; TEXTILE CHEMICALS; PAPER & SURFACE COATINGS
POLYVINYL ALCOHOL
ADHESIVES; TEXTILE CHEMICALS; COATINGS; EMULSIFIERS; SAFETY GLASS
POLYVINYL CHLORIDE-ACETATE COPOLYMERS
PHONOGRAPH RECORDS; FLOOR TILES; SOLUTION COATINGS; PVC PIPE
CELLULOSE ACETATE
FIBERS; CIGARETTE FILTERS; PLASTICS
ACETYL SALICYLIC ACID
ASPIRIN
ACETATE ESTERS
SOLVENTS
CELLULOSE ETHERS
DETERGENT PROMOTERS; TEXTILE CHEMICALS
HERBICIDES
ALKYD RESINS; SYNTHETIC LUBRICANTS
BLEACHING AGENT; CATALYST
POLYVINYL CHLORIDE
PIPE & TUBING; SHEET; CONSTRUCTION MATERIALS; TEXTILE & PAPER COATINGS; FILM; INSULATION & PACKAGING MATERIALS
TRICHLOROETHYLENE
METAL DEGREASING SOLVENT
PERCHLOROETHYLENE
DRY CLEANING & METAL CLEANING SOLVENT
METHYL CHLOROFORM
METAL DEGREASING & CLEANING SOLVENT
POLYVINYLIDENE CHLORIDE & COPOLYMERS
FILM; COATINGS FOR FOOD PACKAGING
CARBAMATE FUNGICIDES; CHELATING AGENT
TETRA ETHYL LEAD
GASOLINE ADDITIVE
REFRIGERANT; SOLVENT; ETHYLATION AGENT
FLAME RETARDANT FOR FIBERS & PLASTICS
LEAD SCAVENGING AGENT
ETHYLATION AGENT; SOLVENT; REFRIGERANT
POLYSTYRENE-GP, HI, EXPANDED
PACKAGING; HOUSEWARE; APPLIANCES; TV CABINETS; FURNITURE; LIGHTING; CONSTRUCTION; TOYS
ACRYLONITRILE-BUTADIENE-STYRENE COPOLYMERS
PIPE & FITTINGS; APPLIANCES; AUTOMOTIVE USE; BUSINESS MACHINES; TELEPHONES; ELECTRICAL & ELECTRONIC APPLICATION
STYRENE-BUTADIENE LATICES
TEXTILE & PAPER COATINGS; EMULSION PAINT; ADHESIVE FOAM PRODUCTS
STYRENE-ACRYLONITRILE COPOLYMERS
APPLIANCES; AUTOMOTIVE USE; HOUSEWARE; FURNITURE; PACKAGING
STYRENE-BUTADIENE ELASTOMERS
TIRES; MECHANICAL RUBBER GOODS; FOOTWEAR; ADHESIVES; CHEWING GUM
UNSATURATED POLYESTERS
REINFORCED: BOATS, BATHTUBS, TANKS, PANELS, TRAYS NON-REINFORCED: DECORATIVE & ELECTRICAL APPLICATIONS
UNSATURATED POLYESTERS
COATING RESINS; ELECTRICAL INSULATION
DYES; PHARMACEUTICALS; PESTICIDES
ETHYLENE-PROPYLENE TERPOLYMERS
AUTOMOTIVE PARTS; TIRES; HOSE & TUBING; WIRE & CABLE
REACTION PROMOTORS, INITIATORS, CATALYSTS
CORROSION INHIBITOR; RUBBER CHEMICALS; INSECTICIDES
SINGLE CELL PROTEIN-YEAST
ANIMAL FEED
TOILETRIES; COSMETICS; INDUSTRIAL SOLVENTS & THINNERS; DETERGENTS; FLAVORS; DISINFECTANTS
SEE ABOVE
ETHYL ACETATE
SOLVENT
SOLVENT; MEDICINALS; PHARMACEUTICALS
PLASTICIZERS; SURFACTANTS
PLASTICIZERS; SURFACTANTS
GRAIN PRESERVATIVES; CELLULOSE PLASTICS; HERBICIDES
HERBICIDES; SOLVENT
RIPING AGENT FOR FRUIT & VEGETABLES

end uses (reprinted from Kniel et al. 1980, pp. 34-35, by courtesy of

Table 4-6. Production emission factors.

Feedstock	% total capacity	Volatile organic compounds (VOC)[α] (kg VOC.t^{-1} C_2H_4 produced)			Ethylene (kg C_2H_4.t^{-1} C_2H_4 produced)		
		Process	Fugitive	Storage	Process[β]	Fugitive[β]	Storage
100% ethane	36	4.3	3.6	---			
100% naphtha	30	7.0	1.5	1.1			
50% naphtha/ 50% gas oil	15	7.0	1.5	1.1			
50% ethane/ 50% propane	19	4.8	3.5	0.1			
Weighted average		5.6	2.6	0.5	0.8	0.4	0.3

[α] U.S. EPA (1980a).

[β] Based on the assumption that 15% of the VOC emissions are in the form of ethylene.

Table 4-7. Ethylene-derived petrochemicals.

Use	1980 Production (10^3 Mg)	Emission factors[α] VOC ($kg.Mg^{-1}$)	Emission factors[α] Ethylene ($kg.Mg^{-1}$)
Polyethylene	615.5	10.0	10.0
Ethylene dichloride	615.0	23.3	12.1
Ethylbenzene	338.0	4.8	N/A[β]
Ethylene oxide	160.5	66.6	56.6
Other	99.5	---	---

α Emission factors taken from U.S. EPA (1980a), Vols. 6, 8, 9.

β Not applicable.

Various kinds of fruits respond differently to exogenous ethylene in the effective times of application, the effective concentrations of ethylene, and the reversibility of the stimulation of respiration by ethylene application (Biale and Young 1981). For bananas, the onset of the climacteric and ripening is normally hastened by 0.11 $mg.m^{-3}$ of ethylene; maximum acceleration is achieved with 1.1 $mg.m^{-3}$. The use of ethylene gas is not regulated in Canada, and the amounts used for fruit ripening or, in the other current major application, for color development and reduction of the curing time for tobacco, are not known.

While the best-known agricultural use of ethylene is in fruit ripening, there is an increasing number of other applications in agriculture and forestry. Ethephon (also called Ethrel, Cepha, Florel), a chemical ($Cl-CH_2-CH_2-PO_3H_2$) that liberates ethylene, is now the most widely used growth regulator in agriculture (Morgan 1982). Etacelasil ($Cl-CH_2-CH_2-Si(O-CH_2-CH_2-O-CH_3)_3$, HOL 1274 ($Cl-CH_2-CH_2-SO_2-CH_2OH$) and HOL 1302 ($Cl-CH_2-CH_2-SO-OH$) also spontaneously release ethylene on contact with water (Lurssen 1982). These chemicals are used worldwide and are applied to almost every phase of plant development. Morgan (1982) and Lurssen (1982) detail current uses, and new applications are continually emerging. An extensive usage of ethylene, or ethylene-producing compounds, for the prevention of lodging in cereals (de Wilde and Furrer 1977; Squires et al. 1980; Wooley 1980; Hill et al. 1982; Smid and Bodnar 1983) is on the horizon. Ethylene also holds promise for weed control through its use for promotion of synchronous germination of certain weed seeds, thus enabling greatly reduced applications of weed control measures (Egley 1982). Such applications for cereals and for weeds have the potential for greatly increasing agricultural usage of ethylene in Canada.

In Canada, Ethephon is registered for use on apples, blueberries, cherries, tomatoes and tobacco. Uses claims of Ethephon acceptable for registration in Canada are: for apples, to promote early red coloring and apple ripening without loosening, to increase flowering of young trees, and to loosen processing apples for easier harvesting; for blueberries, to serve as a coloring and ripening agent for highbush blueberries, and to control black barrenberry for highbush and lowbush blueberries; for sweet or sour cherries, to loosen fruit for easier harvest; for tomatoes, to accelerate ripening; and for tobacco, to promote color development and reduce curing time.

In terms of the active ingredient, the amounts of Ethephon currently used per annum and the major areas in which it is used are: 5040 kg for tobacco, in Ontario; 720 kg for apples, mostly in Ontario and British Columbia, with a little in Nova Scotia; 480 kg for cherries, two-thirds in Ontario and one-third in British Columbia; 4-5 kg for blueberries, in Nova Scotia; total: 9845 $kg.a^{-1}$ (Chang 1983).

Ethephon residue tolerances of the plants, established in Canada by Health and Welfare Canada under the authority of the Food and

Drugs Act, are shown in Table 4-8. A metabolite of Ethephon has been identified as β-D-glucopyranose-1-(2-chloroethyl)phosphonate (Audley 1979).

There do not appear to be any reports on the degree of pollution of the atmosphere resulting from agricultural applications of ethylene-producing chemicals. Possible health hazards resulting from the use of ethylene are discussed in Section 7.3.

4.2.4 EMISSIONS FROM TOBACCO SMOKE

According to Osborne et al. (1956), the volumes of methane and ethylene are 10-20% and about 1.5%, respectively, of the volume of CO in tobacco smoke. Assuming equal distribution between sidestream and mainstream smoke, this means that approximately 1 mg of ethylene is produced per cigarette smoked. In other words, a smoker of 10 cigarettes per day would add about 4 $g.a^{-1}$ ethylene to the environment. The amount of ethylene emitted per cigarette may be an underestimate in view of observations of a higher ratio of ethylene/methane in tobacco smoke (First 1984). It is not known what the total emissions to the indoor and outdoor environments in Canada on an annual basis would be from this source.

4.2.5 SUMMARY

Table 4-9 summarizes the estimated ethylene emissions from anthropogenic sources in Canada, for the base year 1980.

Table 4-8. Ethephon residue tolerances of plants in Canada.

Fruit	Ethephon maximum residue limit ($mg.kg^{-1}$)
Blackberries	20.0
Blueberries	10.0
Sweet cherries	8.0
Apples, figs	3.0
Cranberries, sour cherries and tomatoes	2.0
Citrus fruit	1.0
Cantaloupes, pineapples	0.5

Table 4-9. Estimated Canadian anthropogenic ethylene emissions, 1980.

Category	Source	Ethylene emissions (Mg)
Industrial production processes[α]		25 000
	Ethylene production	1 800
	Ethylene-derived petrochemicals	23 200
Transportation		85 400
	Gasoline engines	68 800
	Diesel engines	15 500
	Aircraft	1 100
Fuel combustion		8 840
	Diesel	170
	Fuelwood	4 800
	Wood waste	3 800
	Incineration	70
Miscellaneous		1 870
	Gasoline marketing	70
	Coke manufacturing	500
	Petroleum refining	1 300
Agriculture and forestry	Application of ethylene and ethylene-releasing chemicals	nd[β]
Total		121 110

[α] These are "uncontrolled" emissions.

[β] Not determined.

5.0 LEVELS IN THE ENVIRONMENT

5.1 AIR

In general, low levels of ethylene in air are associated with rural areas and small towns, whereas high levels are associated with cities and proximity to industrial areas. Location is, however, only one of several factors that influence the atmospheric concentrations of ethylene; time of day, time of year, and meteorological conditions are also important. Diurnal variations have been observed which correlate closely with traffic patterns (Scott et al. 1957; Altshuller and Bellar 1963; Menzies and Shumate 1976) and with the metabolic activity of vegetation in the area (Reid and Watson 1981). Temperature inversions can lead to abnormally high ethylene levels; at night a layering of cooler air at ground level can trap the gas. Byrne (1960, cited by Hasek et al. 1969) reported that ethylene levels were highest on clear, cold nights but were low in windy or rainy weather. Ethylene accumulation is often more pronounced at night in the winter than in the summer, possibly because of radiation inversions (James 1963, cited by Hasek et al. 1969).

Most measurements of levels of ethylene in the air have been made in American, European or Asian cities, where the large number of automobiles or the proximity to industries has resulted in elevated ambient concentrations of ethylene (Lonneman 1977). Some typical values are shown in Table 5-1.

The only published measurements of ethylene levels in Canada were made by Reid and Watson (1981) in southern Alberta (Tables 5-2, 5-3 and 5-4), by the Lambton Industrial Society (1983) in Sarnia (Figure 5-1), and by the Atmospheric Environment Service during plume chemistry studies in the Athabasca Tar Sands region (Bottenheim 1984) and in the vicinity of the coal-fired power plant at Nanticoke, Ontario (Brice and Bottenheim 1983). Substantially lower concentrations of ethylene were observed in these Environment Canada measurements of plume and background air, trace to 15.5 $\mu g.m^{-3}$ (13.5 ppb) (n = 44) and 0.05-1.2 $\mu g.m^{-3}$ (0.04-1.0 ppb) (n = 12), respectively. Because of the scarcity of ambient ethylene measurements in Canadian cities, it can only be assumed that the concentrations are similar to those in cities in the United States of comparable size and population.

The concentration of ethylene in indoor air where smoking has occurred has not been measured. However, it can be estimated to range between 17 and 600 $\mu g.m^{-3}$ (15 and 500 ppb), based on the observations that the volume of ethylene is about 1.5% of the volume of CO in tobacco smoke (Osborne et al. 1956) and that concentrations of CO in rooms where smoking has occurred range between 1 and 35 $mg.m^{-3}$ (1 and 30 ppm) (e.g. Sterling and Kobayashi 1977).

Table 5-1. Selected ambient ethylene concentrations.

Concentration		Location	Reference
$\mu g.m^{-3}$	ppb		
50-60	40-50	Los Angeles	Altshuller and Bellar (1963)
21-24	18-21	Azusa, California	Altshuller et al. (1971)
29-89	25-77	downtown Los Angeles	Altshuller et al. (1971)
40	35	San Francisco Bay area	Stephens et al. (1967)
5.8-37	5-32	Pasadena, California	Menzies and Shumate (1976)
45	40	near Beltsville, Maryland	Abeles and Heggestad (1973)
800	700	center of Washington, D.C.	Abeles and Heggestad (1973)
1-6	0.9-5	Frederick, Maryland	Abeles et al. (1971)
7-8200	6.1-7100	Houston, Texas, area	Gordon and Meeks (1977)
17-25	15-22	Delft, The Netherlands	Guicherit (1975)
18	15.5	Delft, The Netherlands	Bos et al. (1977)
35-58	30-50	Hamburg, West Germany, downwind of an industrial area	Nassar and Goldbach (1979)
~31	~27	Trombay, Bombay, India, in winter, near two major oil refineries	Mohan Rao et al. (1983)
2.0-43.7	1.7-38.0	Trombay, Bombay, daily and monthly variations	Netravalkar and Mohan Rao (1984)
320-470	280-410	Swansea, Wales, near a main road	Harbourn and McCambley (1973)
12-92	10-80	France, within 1 km of an oil refinery	Latarjet et al. (1981)
0.53-11.5	0.46-10	UK, in a rural area over a 2-year period	Cox et al. (1976) (cited by IARC 1979)

Table 5-2. Typical levels of atmospheric ethylene in Calgary, Alberta, at midday (from Reid and Watson 1981).

Location	Concentration	
	$\mu g.m^{-3}$	ppb
Natural wilderness area away from automobiles and industry	14	12
Agricultural land up to western edge of city	14	12
Western half of city	14-28	12-24
City center, industrial area, eastern half	55	48
Agricultural area, city boundary up to 17 km east	55-69	48-60
Agricultural land at least 17 km east of city boundary	14-28	12-24

Table 5-3. Ethylene levels 2 m above ground level, 100 m upwind of a lightly used road, 400 m from the nearest building on the western boundary of the University of Calgary campus[α] (from Reid and Watson 1981).

Date (1981)	Concentration of ethylene		Comments
	$\mu g.m^{-3}$	ppb	
27 Jan	113	98	highly polluted day with an inversion layer
10 Feb	20	17	
13	30	26	
16	25	22	
17	33	29	
18	14	12	(63 $\mu g.m^{-3}$ (55 ppb) in downtown Calgary)
12 May	31	27	
13	23	20	

[α] All measurements were taken at 0800 hours.

Table 5-4. Ethylene levels 2 m above ground level, 72 km northwest of Calgary in a natural subalpine coniferous forest[a] (from Reid and Watson 1981).

Time of day (hours)	Concentration of ethylene	
	$\mu g.m^{-3}$	ppb
0050	4.1	3.6
0900	4.7	4.1
1100	10.2	8.9
1300	14.0	12.2
1500	7.2	6.3
1700	4.5	3.9
1900	4.0	3.5
2100	2.8	2.4
2300	4.1	3.6

[a] 200 m west of Ghost River Campground, upwind of road and human activity, June 1981.

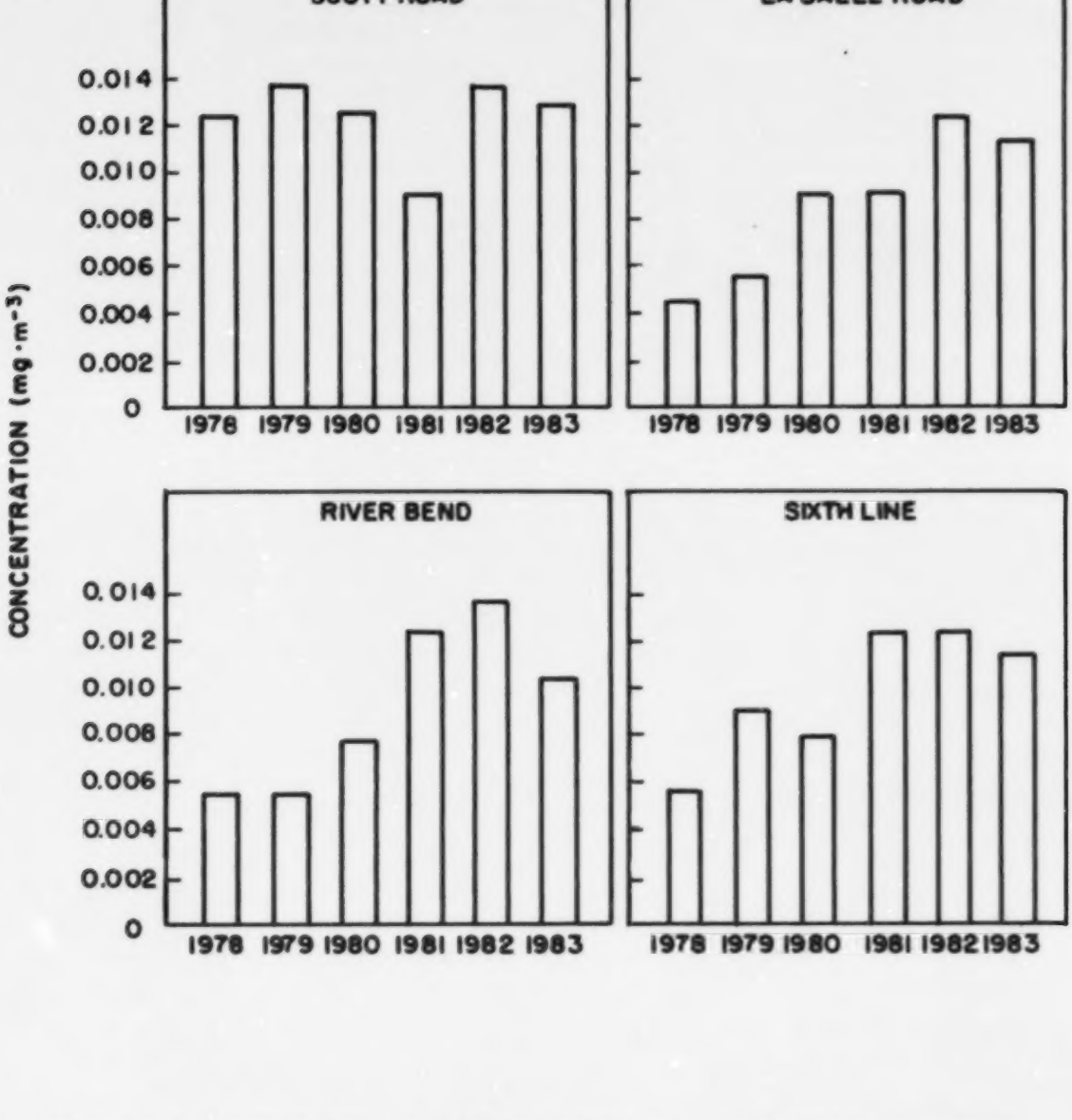


Figure 5-1. Annual average ethylene levels at four locations in Sarnia, 1978-1983 (from Lambton Industrial Society 1983).

5.2 SOIL

Smith and Russell (1969) reported that the gas phase of anaerobic soils contained about 12 $mg.m^{-3}$ (10 ppm) ethylene. In soils exposed to air, on the other hand, levels ranged from 0.08 $mg.m^{-3}$ (0.07 ppm) at 15 cm below the surface to 0.16 $mg.m^{-3}$ (0.14 ppm) at a depth of 60 cm. The authors concluded that high concentrations of ethylene were unlikely in well aerated soils, a finding confirmed by Smith and Dowdell (1974). In waterlogged soils, concentrations of ethylene in air at equilibrium with the water in the soil were calculated to be 14 and 23 $mg.m^{-3}$ (12 and 20 ppm) for two soils tested (Smith and Russell 1969). Campbell and Moreau (1979) also demonstrated that ethylene levels were high in waterlogged soils. Dowdell et al. (1972) found that ethylene concentrations in waterlogged clay soils were highest in the winter and lowest in the spring.

5.3 WATER

Ethylene has been measured in the Gulf of Mexico (1.7-35.0 $nL.L^{-1}$ (ppb); 1 nL = 40.8 pmol), the Caribbean Sea (2.2-12.0 $nL.L^{-1}$ (ppb)), the Atlantic Ocean (0.7-30.0 $nL.L^{-1}$ (ppb)) and the Pacific Ocean (2-11 $nL.L^{-1}$ (ppb)) (Swinnerton and Lamontagne 1974; Lamontagne et al. 1975). Concentrations were relatively uniform in the open ocean and near shore; they generally decreased with increasing depth in the water column and reached trace levels at 150-200 m. Maximum concentrations, however, were often found in water depths of 30-100 m (Swinnerton and Lamontagne 1974). Production appeared to be related to biological processes or photochemical reactions with organic matter in seawater. McAuliffe (1976) calculated the total quantity of ethylene and propylene in the surface 200 m of the North Atlantic to be 620 000 t; both unsaturated compounds appeared to be primarily biogenic in origin.

5.4 PLANTS

The internal level of ethylene in plants increases as the rate of ethylene production increases; the relationship between these two parameters is defined as the conversion constant in parts per million of ethylene in the tissue per microlitre of ethylene produced per kilogram fresh weight per hour (Table 5-5) (Abeles 1973). The internal levels therefore depend on many factors that affect the rate of production, e.g. organ (surface-to-volume ratio, epidermal anatomy) and stage of development. In general, fruits have higher conversion constants than does vegetative tissue. In addition, exogenous ethylene will cause the internal levels to increase (see Section 6.2.2.1).

Table 5-5. Internal levels of ethylene in plant tissues and conversion constants (after Abeles 1973)[α].

Tissue	Internal levels (ppm)	Conversion constant[β] (ppm.μL^{-1} ethylene. kg^{-1}.h^{-1})
Fruit		
Apple (McIntosh and Baldwin)	25.0-2500	2.0-13
Avocado (Choquette)	29.0-74.0	2.2
Banana (Gros Michel)	0.05-2.1	1.9
Banana	0.01	1.0
Cantaloupe	1.0-16	2.5
Lemon	0.11-0.17	1.8
Lime	0.3-1.96	3.4
Mango	0.04-3.0	3.8
Nectarine	3.6-602	13.0
Orange (Navel)	0.1-1.0	0.5-1
Orange (Valencia)	0.13-0.32	4.0
Passion fruit	466-530	9.0
Peach (Elberta)	0.9-20.7	5.2
Pear (Bosc)	80	5.0
Pineapple	0.16-0.40	2.0-7.0
Pineapple	80-1140	3000
Plum	0.14-0.23	3.9
Summer squash	0.04-2.1	1.9
Tomato	3.6-30	3.2-4.4
Tomato	-[γ]	3.0
Leaf		
Cotton	0.25-0.75	0.25-0.34
Huisache	-	0.024
Root		
Pea	2.0	0.3
Pea	-	0.16
Stem		
Bean	0.8	0.42
Pea	-	0.13

NOTE: Units are as shown in original table. 1 ppm = 1.15 mg.m^{-3} ethylene. 1 μL = 40.8 nmol ethylene.

[α] Original references are given in Abeles (1973).

[β] Conversion constant = internal level/rate of production, expressed as parts per million of ethylene in the plant tissue per microlitre of ethylene produced per kilogram fresh weight per hour.

[γ] Data not available.

5.5 HUMANS

Conkle *et al.* (1975) detected the exhalation of ethylene of metabolic origin at rates of 0.91 $\mu g.h^{-1}$ (nonsmoker) and 120 $\mu g.h^{-1}$ (smoker) in 2 of 10 human subjects. Chandra and Spencer (1963a) found that the ethylene content of exhaled air of a normal adult subject was nearly double that of the inhaled air (7.8 as opposed to 4.0 $\mu g.m^{-3}$ (6.8 versus 3.5 ppb)).

6.0 ENVIRONMENTAL DYNAMICS

6.1 ABIOTIC FATE

Once released into the atmosphere, ethylene, like other low-molecular-weight gases, is dispersed by turbulent processes; this dispersion is accompanied by chemical transformations that decrease ambient and ground level concentrations. The major processes for the loss of ethylene from the atmosphere are reactions with photochemical oxidants and uptake by soil microorganisms (see Section 6.2.1).

Ethylene, being an unsaturated, vapor-phase hydrocarbon, may participate in photochemical reactions in the atmosphere under certain conditions. The two principal chemical reactions governing the fate of ethylene in air are (i) reaction with ozone and (ii) reaction with the hydroxyl radical. The chemical mechanism involved in ozone formation in a nitrogen oxides - hydrocarbon polluted atmosphere is relatively well understood (cf. Leighton 1961; Kerr et al. 1972; Butler 1979; Calvert 1982; Niki et al. 1983) and may be illustrated as follows:

$$NO_2 + h\nu \rightarrow O[^3P] + NO \qquad \lambda = <340 \text{ nm} \qquad (1)$$

$$O[^3P] + O_2 + M \rightarrow O_3 + M \qquad (2)$$

$$O_3 + NO \rightarrow O_2 + NO_2 \qquad (3)$$

$$O_3 + NO_2 \rightarrow O_2 + NO_3 \qquad (4)$$

$$O_3 + \text{Alkene} \rightarrow \text{Products} \qquad (5)$$

$$O_3 + h\nu \rightarrow O[^1D, {}^3P] + O_2[{}^1\Delta_g, {}^3\Sigma_g^+] \qquad \lambda = <310 \text{ nm} \qquad (6)$$

Reactions (1) through (6) are considered to be the primary ones governing the concentration of ozone in a polluted, irradiated air mass in the absence of significant atmospheric turbulence. In the atmosphere of typical photochemical smog episodes, the reaction of ozone with alkenes (reaction (5)) is slower than its reaction with nitric oxide (reaction (3)). Numerous studies have been carried out to determine the rate of reaction of ozone with alkenes. From those studies conducted at or near 293 K and 101.3 kPa (Japar et al. 1974; Su et al. 1980; Adeniji et al. 1981; Kan et al. 1981; Niki et al. 1981; Atkinson et al. 1982), the reaction of ozone with ethylene is considered to be bimolecular, with a rate constant of 1.8×10^{-18} $cm^3.s^{-1}$ per molecule.

Ozone attack on ethylene follows complex degradation pathways, and gives rise to numerous products such as ozonides, aldehydes, carbon monoxide, carbon dioxide and acids (see Figure 6-1). Sulfur dioxide may also be oxidized to sulfates (Su et al. 1980; Calvert 1982; Niki et al. 1983).

The reaction of the hydroxyl radical (HO•) with ethylene is initiated by the electrophilic addition of the radical to the ethylene double bond; subsequent radical chain reactions lead to the production

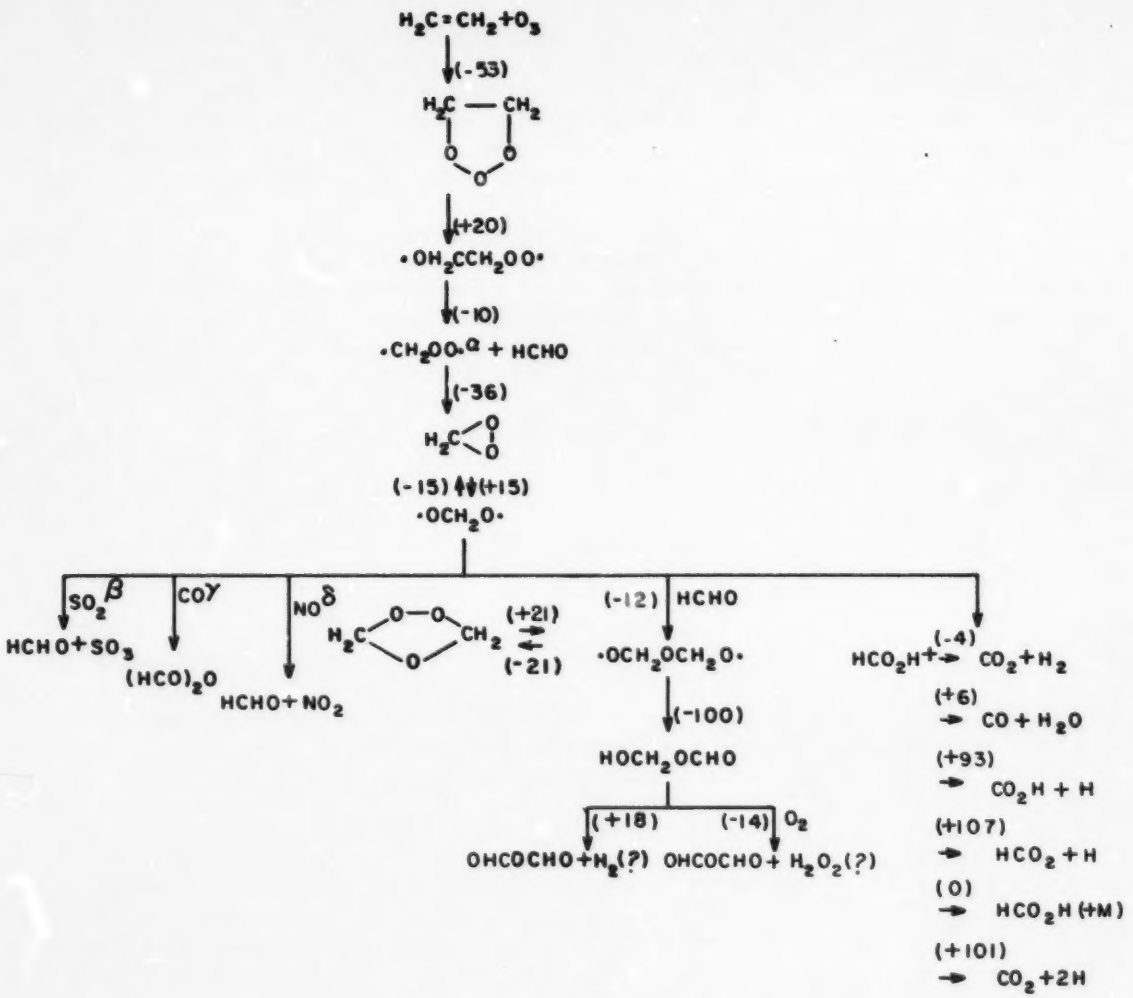


Figure 6-1. Reaction scheme for the oxidation of ethylene in the presence of ozone. α, β, γ, δ refer to the relative rates of reaction k_α: k_β: k_γ = 100:400:0.7 (k_δ unknown). ΔH values are in brackets (from Calvert 1982).

of aldehydes, acids and nitrogen dioxide (in an ethylene-NO system) (Tully 1983). The currently accepted reaction scheme in the presence of nitrous oxide is illustrated in Figure 6-2. The stoichiometry for the overall reaction is:

$$(HO\cdot) + C_2H_4 + 2NO + 2O_2 \rightarrow 2HCHO + 2NO_2 + (HO\cdot) \qquad (7)$$

Ethylene is converted almost quantitatively to formaldehyde (HCHO) (i.e. $\Delta HCHO/\Delta C_2H_4 \approx 2$). In addition, for each ethylene molecule consumed, two molecules of nitrogen dioxide are formed. Small amounts (≈3%) of formic acid (HCOOH) are also formed. The initial reaction of hydroxyl radical with ethylene is considered to be bimolecular; the rate constant is about 8.3×10^{-12} $cm^{-3}.s^{-1}$ per molecule at standard pressure and temperature (Cox 1975; Lloyd et al. 1976).

Assuming that the initial concentrations of reactants are approximately equal and that bimolecular kinetics apply, the lifetime of ethylene in air in the presence of ozone or hydroxyl radical can be estimated. For an ozone concentration of 1×10^{12} molecules per cubic centimetre (usually considered to be the background concentration (Altshuller 1980)), the rate of reaction of ethylene and ozone is about 1.8×10^{-6} s^{-1}. The hydroxyl radical concentration at 50-60°N latitude is less than 1×10^{4} molecules per cubic centimetre in winter (January) and 1.8×10^{5} molecules per cubic centimetre in summer (July) (Bottenheim and Strausz 1980). For a hydroxyl radical concentration of 1×10^{5} molecules per cubic centimetre, the rate of the bimolecular reaction between ethylene and hydroxyl radical is approximately 0.8×10^{-6} s^{-1}. Under these conditions, the residence time of ethylene in air is approximately 6.5 d in the presence of ozone and 14 d in the presence of hydroxyl radicals. Residence times will decrease with an increase in oxidant concentration.

It appears that ethylene is one of the least photochemically reactive compounds of the vapor-phase, unsaturated, nonaromatic hydrocarbons. For example, the concentrations of propene and butene in automobile exhaust are one-half and one-eighth the concentration of ethylene (U.S. NAS 1976). However, propene and butene react seven and six times faster than ethylene with ozone (Japar et al. 1974). Of a series of 15 vapor-phase, acyclic and cyclic olefins, ethylene was the least reactive in the presence of ozone. With respect to reaction with hydroxyl radical, propene and butene react three and eight times faster than does ethylene (Lloyd et al. 1976).

6.2 BIOTIC FATE

6.2.1 SOIL MICROORGANISMS

Little is known about the metabolism of ethylene by soil microorganisms, but the process of ethylene degradation is significant

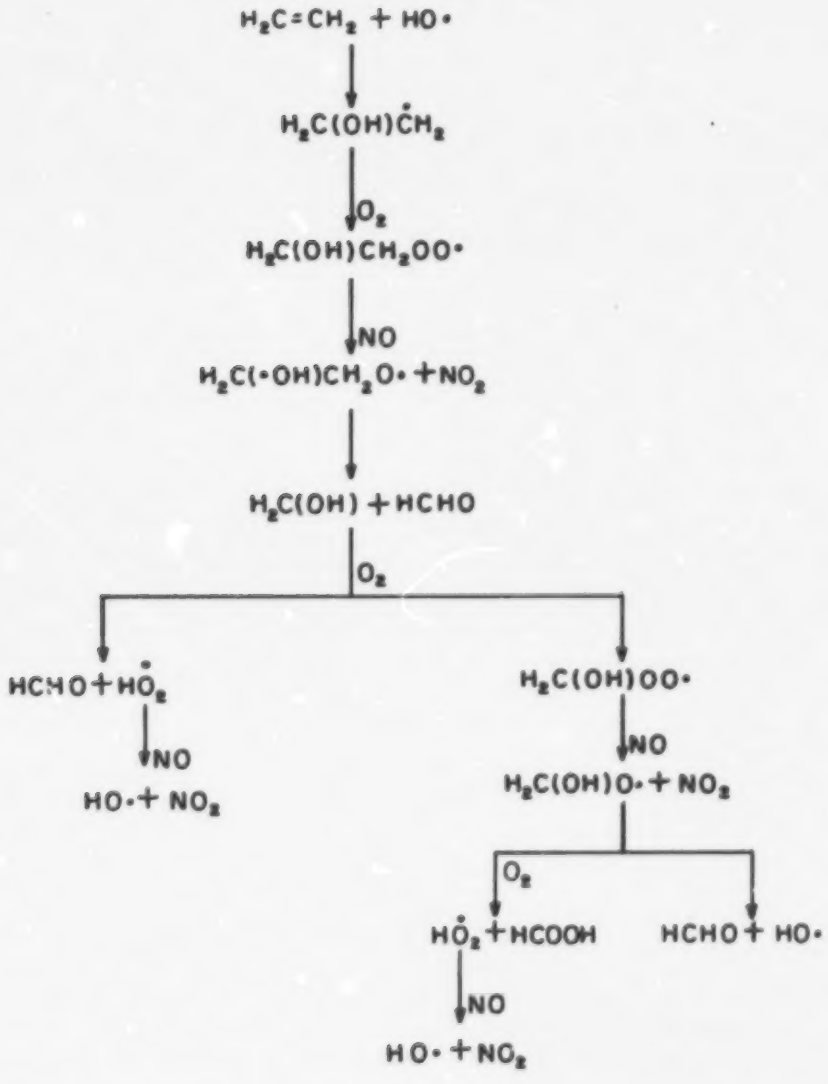


Figure 6-2. Reaction scheme for the oxidation of ethylene initiated by hydroxyl radical in the presence of nitrous oxide (from Niki et al. 1981).

when one considers that an estimated 7 x 10^6 t of ethylene, one-half of the yearly ethylene production in the United States, is degraded microbiologically in the soil each year (Abeles et al. 1971).

Because the soil is both a source of and a sink for ethylene, most laboratory studies measuring ethylene in the headspace actually measure accumulation and not production, since consumption of ethylene occurs simultaneously with production. Therefore, ethylene accumulation over time in closed vials and flasks reflects both the increase from producers as a result of oxygen reduction and a decrease in consumption by ethylene consumers.

Cornforth (1975) showed that ethylene decomposition was almost completely inhibited in autoclaved soils whereas ethylene consumption was substantial in control soils. However, the decomposition rate of ethylene increased with each addition of ethylene into the flasks, suggesting an increase in the responsive population. The rate of CO_2 production decreased with each introduction of ethylene. The decomposition of ethylene was most rapid in moist soil, and slowest in oven-dried soil, in which over 90% remained after 16 d. Ethylene decomposition was slower in air-dried and frozen soil than in fresh soil. Long-term storage in the air-dried state reduced the ethylene decomposition rate. The rate of ethylene decomposition was directly related to O_2 concentration. Ethylene was decomposed in flasks containing 0.5% O_2 until the O_2 concentration was reduced to less than 0.2%. The results suggested that the ethylene decomposers are aerobes and that they decompose ethylene approximately 50 times faster than it is produced in anaerobic soil.

The addition of glucose to soil slowed the decomposition of added ethylene, but stimulated production of carbon dioxide (Cornforth 1975). Ethylene had no effect on CO_2 production. Addition of CO_2 did slow the decomposition of ethylene, a process that may influence its accumulation in anaerobic soil zones. However, since the rate of decomposition is much greater than the rate of production, CO_2 will have little effect on narrowing the ratio of anaerobic to aerobic soil volumes necessary for ethylene to accumulate. A small group of aerobic organisms was believed to be decomposing ethylene in the soil.

In rice culture, Yoshida and Suzuki (1975) reported toxic levels of ethylene accumulation in submerged rice soils. However, substantial oxidation of ethylene was shown to occur in the rice rhizosphere, but not in the rhizosphere of upland crops and upland rice. Populations of certain soil microflora able to degrade ethylene may accumulate under submerged soil in association with rice.

Abeles et al. (1971) showed that the removal of atmospheric ethylene, sulfur dioxide, and nitrogen dioxide by soil was biological in nature and considerably greater than the predicted rates of production. Autoclaving reduced the uptake of ethylene by soil. Sorption by soil

colloids would also reduce the apparent levels in the soil atmosphere. Reducing the oxygen level in the soil atmosphere to 1% reduced the ethylene uptake by half. A lag in ethylene uptake was observed which was reduced with subsequent additions.

Abeles (1983, 1984a) found that Mycobacterium paraffinicum exhibited a linear rate of ethylene oxidation that correlated linearly with concentration of ethylene from 0.9 to 900 $mg.m^{-3}$ (1-1000 ppm). CO_2 had no effect on ethylene oxidation by this organism.

The biological oxidation of ethylene was studied by de Bont (1976) who isolated five strains of immotile, gram-positive, acid-fast, pleomorphic organisms, resembling the genus Mycobacterium. These ethylene-oxidizing organisms grew well on saturated alkanes and on ordinary carbon sources. Ethylene itself served to induce its uptake by these organisms. The fact that these organisms were able to oxidize ethylene at such low levels is significant in terms of the low values reported to damage plant roots (Abeles et al. 1971).

The catabolic pathway of ethylene in Mycobacterium E20 is possibly the most well understood of such pathways in soil microorganisms at this time (de Bont and Harder 1978). Studies on ethylene metabolism by de Bont and Albers (1976) showed that ethylene oxide was a product of ethylene catabolism by strain E20. From the specific activities of isocitrate lyase and malate synthetase in cell-free extracts, the glyoxylate cycle was implicated in ethylene metabolism, indicating that acetyl-CoA is a metabolite of ethylene catabolism. Ethylene glycol was not formed from ethylene oxide. Because the strains tested were able to grow on ethanolamine, the authors speculated that ethanolamine was a product formed by the ring-opening of the epoxide with ammonia.

Later studies, however, showed that ethylene was oxidized to ethylene oxide by an oxidizing enzyme system utilizing a monooxygenase (de Bont et al. 1979). The enzyme derives its oxygen from molecular oxygen and requires NAD^+, acetyl-CoA, and an unknown, heat-stable, dialyzable cofactor (de Bont and Harder 1978). The 1-alkene monooxygenase involved in ethylene catabolism is specific and differs from the n-alkane monooxygenases (de Bont et al. 1979). Ethylene oxide was shown not to yield ethylene glycol by hydrolysis. The glycerate pathway was not active because of inactivity of the key enzyme, glyoxylate carboligase. The ethylene oxide intermediate was suggested to be an end product of the catabolic sequence that feeds into the citric acid cycle via acetyl-CoA; it plays a role similar to that of acetate in ethane catabolism.

A nitrogen-fixing methane-oxidizing bacterium from soil was reported by de Bont and Mulder (1974) to co-oxidize ethylene when growing on methane. In the presence of acetylene used in the acetylene-reduction assay test for nitrogenase activity, acetylene

reduction (but not ethylene production) was reported only when bacteria were grown on methanol, but not on methane, although $^{15}N_2$ was fixed.

6.2.2 PLANTS

6.2.2.1 Uptake and Diffusion

It is generally accepted that the primary route of entry for gaseous pollutants into higher plants is through the stomata (Mukammal 1976). Although it has not been experimentally proven, this is assumed to hold true for ethylene, which could enter through the epidermis of the stem, petiole or leaf blade (Zimmerman et al. 1931). The ease of uptake of ethylene into higher plants would therefore depend to some extent on the number and size of the stomatal openings; ethylene would enter leaves, for example, which are assimilatory structures and hence have great porosity (i.e. more and larger stomata), more readily than, for example, fruits which are modified to minimize water loss (Abeles 1973).

Ethylene itself has been observed to affect the number and size of stomatal openings in plant tissue. For example, ethylene caused changes in the ratio of epidermal cells to stomata in developing sunflower (Helianthus anuus) leaves, increasing it on the upper surface and decreasing it on the lower surface (Funke et al. 1938, cited by Abeles 1973). In addition, ethylene has been reported to influence the degree of stomatal opening, the exact response (i.e. no effect, stimulate opening, or stimulate closing) depending on the species tested (e.g. Saad 1953/54, cited by Abeles 1973; Pallaghy and Raschke 1972; Vitagliano and Hoad 1978; Madhavan et al. 1983). Induction of stomatal closure in the presence of ambient ethylene could be interpreted as a defence mechanism, whereas stimulation of stomatal opening would be a liability to the plant since it would promote increased injury. Of course, stomatal closure might also cause an accumulation of ethylene, hence increasing the damage.

The uptake of ethylene into plant tissue would depend not only on the number and size of stomatal openings, but also on the diffusion gradient in the plant, determined by the rate of ethylene production (Burg and Burg 1962), resistance to diffusion, and the surface-to-volume ratio (Abeles 1973). For example, if the gradient between internal and external levels of ethylene were 1 $mg.m^{-3}$ (ppm) in ethylene-free air, then the addition of 0.1 mg.[illegible] ppm) ethylene to the external air would cause internal levels to rise to 1.1 $mg.m^{-3}$ (ppm); the diffusion gradient itself would remain unchanged (Abeles 1973). Cuticular barriers probably maintain internal ethylene concentrations at a substantially higher level than external concentrations (Burg 1968).

After being taken up into the plant through the stomata, the gaseous pollutant must reach the living cells in order to produce

injury. Ethylene, a small molecule, should move readily through plant tissue by diffusive processes, following Fick's law of diffusion (Burg and Burg 1962, 1965b). Its small size, as well as its solubility in both lipophilic and aqueous systems, should facilitate its movement through plants, not only through air spaces but possibly through membranes as well (Leopold and Kriedemann 1975).

The ease of diffusion and the diffusion path for ethylene appear, however, to depend on the tissue involved. Some workers have suggested that ethylene moves readily between different parts of the plant (e.g. Zimmerman et al. 1931; Jackson and Campbell 1975). Zimmerman et al. (1931) observed the movement of ethylene across dead stem tissue, suggesting that movement through the plant is passive. Barmore and Biggs (1970), however, found that various parts of the citrus leaf and fruit offered different degrees of resistance to ethylene diffusion. Zeroni et al. (1977) noted that ethylene did not appear to move between different parts of Vicia faba in physiologically significant amounts; the two main factors that appeared to limit ethylene movement were resistance in the longitudinal direction and loss of ethylene by emanation from the surface of the plant. In wood, on the other hand, ethylene appeared to move 100 times faster longitudinally than horizontally (Elers 1965, cited by Abeles 1973). There is also evidence that under anaerobic, waterlogged conditions, an ethylene precursor, 1-aminocyclopropane-1-carboxylic acid (ACC), is readily transported from the roots to the shoot via the transpiration stream (Bradford and Yang 1980, 1981).

There is considerable controversy regarding the movement of applied Ethephon (which releases ethylene) through plants. Some have found no movement (Hume and Lovell 1983), whereas others have observed ready translocation (Kwong and Lagerstedt 1977).

Although the diffusion of ethylene within plants is not universally accepted, it is generally agreed that some of the gaseous plant hormone can be removed from the plant by diffusion to the external atmosphere (Abeles 1973). Ethylene can exit from fruits, for example, at the point of stem attachment or through the skin (Burg and Burg 1965b; Barmore and Biggs 1970). The gas would exit from leaves primarily through the stomatal openings.

6.2.2.2 Incorporation and Metabolism

Several investigators have examined the incorporation of ethylene from the surrounding atmosphere into plants. Buhler et al. (1957), for example, found that 0.05% of the label in ^{14}C-ethylene was incorporated into the organic acid fraction of avocado and pear tissue. However, because incorporation was so low, and because it did not occur in any other fruits tested (e.g. oranges, apples, tomatoes), the authors concluded that ethylene was probably a terminal product of a sequence of

metabolic processes in fruit and could not be metabolized; the observed incorporation was probably the result of exchange reactions in the biosynthesis of ethylene in fruit. In the experiments of Buhler et al. (1957), no radioactivity was found in the respiratory CO_2 of the exposed pears or avocadoes.

Jansen (1963, 1964) observed that 0.015-0.018 and 0.042-0.043% of ^3H- and ^{14}C-ethylene at 290 and 2300 $mg.m^{-3}$ (250, 2000 ppm), respectively, were incorporated into avocado fruit in a 4-h period. Of the total radioactivity incorporated by ^3H- and ^{14}C-ethylene metabolism (at 2300 $mg.m^{-3}$; 2000 ppm), 12.5 and 0.2%, respectively, were in toluene; 1 and 4%, respectively, were in benzene. The author concluded that in the metabolism of ethylene by the avocado, a considerable portion of the hydrogen is removed from ethylene by a dehydrogenation process, and proceeds along a pathway different from that followed by carbon; part of the hydrogen and carbon are, however, metabolized along the same pathway. Jansen (1964) also found that some of the ethylene was oxidized to CO_2, with more oxidation occurring at the lower ethylene concentration. Shimokawa and Kasai (1968) suggested that ^{14}C-ethylene or a metabolite was incorporated into ribonucleic acid (RNA) in Japanese morning glory, possibly inducing a conformational change in the RNA. Hall et al. (1961, cited by Abeles 1973) found that ^{14}C-ethylene was incorporated in several substances by cotton and Coleus plants.

These and similar experiments have been criticized by several authors (e.g. Abeles 1973; Lieberman 1979), because no precautions were taken to ensure that the label incorporated into plant tissue was from the ethylene itself, rather than from impurities in the radioactive compound used, or to ensure that microbial contamination had not occurred. Jerie and Hall (1978) found it unusual that Jansen (1963, 1964) had observed incorporation of label into such compounds as benzene and toluene, compounds that are unexpected in higher plants. Because of these criticisms and because no incorporation was observed in some fruit tissues (Buhler et al. 1957; Behmer 1958), it was concluded that ethylene was probably not metabolized by plants (e.g. Abeles 1973).

However, more recent studies have shown that this conclusion was incorrect, at least for some plant tissues. Jerie and Hall (1978), for example, concluded that 85-95% of the applied ^{14}C-ethylene was metabolized in less than 2 h to ethylene oxide by mature cotyledons of the broad bean, Vicia faba. No labelled CO_2 was detected in these experiments. The authors proposed that ethylene oxide formation was a normal process, since it occurred at ethylene concentrations similar to those found in tissues of the broad bean. Lieberman (1979) found Jerie and Hall's (1978) report of such high ethylene metabolism unusual, since most previous experiments showed less than 1% uptake and metabolism of applied $^{14}C_2H_4$. Instead, he suggested that the ethylene oxide was produced (i) artifactually within the living cell, (ii) by a non-physiological reaction, (iii) uniquely in Vicia faba, or (iv) by an impurity in the $^{14}C_2H_4$. This latter point is worth emphasizing; the

group led by Hall, which included Jerie and Dodds, used an unpurified ethylene in all its experiments. This must be taken into consideration in the interpretation of the group's results.

Dodds et al. (1979, 1980) confirmed the findings of Jerie and Hall (1978), showing that a small amount of ethylene oxide was produced as a metabolite of ethylene by cell-free preparations from Vicia faba cotyledons. A K_m value of 4.17×10^{-10} M for ethylene was calculated, corresponding to a gaseous concentration of 0.0096 $\mu L.L^{-1}$ (ppm), close to endogenous ethylene concentrations in the air spaces of some plant tissues. The enzyme involved required molecular oxygen, and was inhibited competitively by propylene and other structural analogues of ethylene. No labelled CO_2 was present in these experiments with Vicia faba.

Blomstrom and Beyer (1980) found that two of the products of ethylene metabolism in pea (Pisum sativum) tissue were ethylene glycol and a glucose conjugate. Since ethylene oxide is a likely precursor of ethylene glycol, and since Vicia faba cotyledons rapidly converted ethylene to ethylene oxide (Jerie and Hall 1978; Dodds et al. 1979), the authors suggested that ethylene oxide might escape from Vicia faba cotyledons before undergoing hydrolysis, or that the hydrolysis step might be blocked in this tissue.

Beyer (1981) described experiments using purified ^{14}C-ethylene in which pea (Pisum sativum) tissue was able to convert ^{14}C-ethylene to $^{14}CO_2$ (95% recovery), a finding confirmed by Evans et al. (1984). Because these results contradicted those of Jerie and Hall (1978) and Blomstrom and Beyer (1980), Beyer (1981) repeated his experiments, extending the observation period to 24 h instead of 6 h, in order to see if any ethylene oxide or ethylene glycol was formed by ethylene metabolism in peas. It was discovered that pea seedlings continuously convert ^{14}C-ethylene to $^{14}CO_2$, but that after a lag period of about 7 h, ^{14}C-ethylene glycol is formed by alkaline hydrolysis of ethylene oxide which is derived from ethylene.

Beyer and co-workers determined several characteristics of the observed system of ethylene metabolism in several plant tissues: (i) considerable variation during growth and development, often with an increase with age (Beyer 1975b); (ii) dependence on a high degree of tissue integrity, and inhibition by heat and tissue homogenization, as well as by metabolic inhibitors, anaerobic conditions, and chelating agents (Beyer 1975a, b; Beyer and Blomstrom 1980); (iii) stimulation by a temperature increase from 15 to 25°C, and decrease above 28-30°C (Beyer and Blomstrom 1980); (iv) linear increase with ^{14}C-ethylene concentration (Beyer 1975a, b, 1981), and saturation at a level well above 100 $\mu L.L^{-1}$ (ppm); (v) increase with increasing ambient oxygen concentration in some tissues (Beyer 1975a, b; Beyer and Blomstrom 1980); (vi) inhibition by CO_2 of conversion to CO_2 (Beyer 1975a, b); (vii) no effect of light and other plant hormones (Beyer 1975a, b);

(viii) reduction in intact pea seedlings by the detachment of cotyledons from the root-shoot axis (Beyer 1975a, b, 1981); and (ix) enormous variation in rate in different tissues (Beyer 1981).

Although the formation of metabolites from ethylene has been observed in a number of plant tissues, including Vicia faba cotyledons (Jerie and Hall 1978), pea (Pisum sativum) seedlings (Blomstrom and Beyer 1980), carnations (Dianthus caryophyllus) (Beyer 1977), morning glory (Ipomoea tricolor) flowers (Beyer and Sundin 1978), and tomato fruit (Blomstrom and Beyer 1980), few generalizations can be made about the metabolic process. There are some tissues, including developing cotyledons of Phaseolus vulgaris (Hall et al. 1982a), in which ethylene metabolism does not appear to occur. Even within a species of plant there are discrepancies; the ability of cotyledons of Vicia faba, for example, to metabolize ethylene to ethylene oxide appears to depend on the variety (Dodds et al. 1982/1983). Again it should be pointed out that the use of impure ethylene in the experiments could account for some of these discrepancies.

The formation of $^{14}CO_2$ from ^{14}C-ethylene metabolism has also not been observed by all workers. Although $^{14}CO_2$ production was detected by Jansen (1964), Beyer (1975a, b, 1981), Blomstrom and Beyer (1980) and Evans et al. (1984), labelled carbon dioxide was not observed by Buhler et al. (1957), Jerie and Hall (1978) and Dodds et al. (1979), nor by several workers who applied ^{14}C-Ethephon (which presumably degrades to ethylene) to various plant tissues (mature tobacco leaf discs (Nicotiana tabacum), Domir and Foy 1978a, b; olives (Olea europea), Epstein et al. 1977; citrus fruits, Young and Jahn 1975, cited by Epstein et al. 1977; tomatoes, Yamaguchi et al. 1971).

6.2.2.3 Physiological Significance of Metabolism

Several different groups of workers have speculated on the physiological significance of ethylene metabolism in plant tissue. Jerie and Hall (1978), for example, suggested that its function was related to ontogeny, since ethylene oxide production by Vicia faba cotyledons increased with stage of maturity. Dodds et al. (1982/1983) also observed that the rate of ethylene metabolism in leaf, stem and root tissues of Vicia faba varied during ontogeny.

Dodds et al. (1982/1983), noting a correlation between resistance of Vicia faba cotyledons to waterlogging and the extent of ethylene metabolism in tissues, hypothesized that the resistance was linked to the removal of "stress ethylene" by metabolism to ethylene oxide. Others (e.g. Jerie and Hall 1978) agree that the role of ethylene metabolism may be the regulation of endogenous ethylene levels. This does not, however, appear to be true, since (i) the amount

metabolized represents only a few percent of that naturally produced (Beyer 1981); (ii) there is no increase in the amount of ethylene evolved from tissue when metabolism is inhibited (Beyer 1981); (iii) peak rates in biosynthesis do not always correspond with peak rates in metabolism (Jerie and Hall 1978); and (iv) ethylene diffuses rapidly out of most tissues (Abeles 1973), making an ethylene inactivation system unnecessary (although rapid equilibration may not occur inside the plant cells (Jerie and Hall 1978)).

Instead, Beyer (1975a, b) suggested that ethylene metabolism occurred at the binding site as an integral part of the ethylene action mechanism. Ethylene is known to bind to sites in various plant tissues (e.g. Phaseolus vulgaris cotyledons, Hall et al. 1982a, b). The receptor site has been hypothesized to contain copper, since (i) ethylene can complex with copper; (ii) ethylene metabolism is sensitive to such copper chelators as CS_2 (Beyer 1977; Abeles 1984a); (iii) copper can catalyze the oxidation of ethylene in nonbiological aqueous systems (Blomstrom and Beyer 1980; Beyer 1981); and (iv) Cu(I)-ethylene coordination chemistry is consistent with the proposed role of copper at the ethylene receptor site in plant tissue (Thompson et al. 1983). Burg and Burg (1965a) developed a model in which ethylene was viewed as a dissociable activator, binding to the metal of a metalloenzyme whose reactivity to ethylene was dependent on an interaction with O_2; the binding then triggered a response to ethylene. However, subsequent studies (e.g. Beyer 1972) showed that ethylene did not act as a dissociable activator molecule; it was therefore hypothesized instead that metabolism of ethylene occurred during the course of receptor site activation (Beyer 1981).

Evidence exists to support this proposal. For example, there is a close correlation between changes in ethylene tissue sensitivity and changes in ethylene metabolism (Beyer 1975a, b). The most convincing evidence lies in the anti-ethylene action of CO_2 and silver ions (Ag^+). Ag^+ significantly and quantitatively inhibited the conversion of $^{14}C_2H_4$ to ethylene oxide in pea (Pisum sativum) tissue while not affecting the oxidation of $^{14}C_2H_4$ to $^{14}CO_2$; on the other hand, high CO_2 concentrations inhibited $^{14}C_2H_4$ oxidation to $^{14}CO_2$ without affecting the formation of ethylene metabolites (Beyer 1975a, b, 1979, 1981). In contrast, Hall et al. (1982b) observed that CO_2, while competitively inhibiting conversion of ethylene to CO_2 in Pisum sativum, actually promoted conversion to ethylene oxide by increasing the affinity of the system for ethylene. Abeles (1983) found that CO_2 had no effect on ethylene oxidation by Vicia faba cotyledons. Both Ag^+ and CO_2 significantly inhibited ethylene-induced growth retardation in pea tissue (Beyer 1979).

There appear, therefore, to be two ethylene metabolizing systems in at least some plant tissues. One yields CO_2, and is inhibited by it. The other, originally believed to lead to incorporation of ethylene or its metabolites into soluble tissue

components, is now known to produce ethylene oxide, and is inhibited by Ag^+. Since both inhibitors are known to counteract the effects of ethylene in several systems (e.g. Beyer 1979), it appears that ethylene metabolism is involved in the mediation of ethylene-induced responses. Again, it should be emphasized that this probably holds true for only certain plant species. Abeles (1983, 1984b), for example, concluded that the ethylene monooxygenase activity of Vicia faba cotyledons was not a reflection of hormone action, based on studies of inhibition of the oxidation process.

Although ethylene oxide is further metabolized to ethylene glycol in some tissues, there is possibly a role for ethylene oxide itself in ethylene action, as the epoxide appears to act synergistically with ethylene in the growth inhibition of peas, as well as amplifying other ethylene effects (Beyer 1981, 1984) (see also Section 7.2.5). A tentative outline for the steps involved in ethylene metabolism and action in plant tissue is given in Figure 6-3. An excellent review of the metabolism of ethylene by plants and its physiological significance has recently been published (Beyer 1984).

6.2.3 HUMANS AND OTHER ANIMALS

Because the processes of uptake, retention, metabolism and elimination of ethylene by humans and other animals are closely interconnected, it is difficult, if not impossible, to completely separate the results of studies concerning these processes. For the purposes of this discussion, a more useful division appears to be between those studies involving simple exposures of humans and other animals to ethylene and those involving animals exposed to ethylene following pretreatment with some chemical to induce the hepatic mixed function oxidase system.

6.2.3.1 Simple Ethylene Exposure

Andersen et al. (1980) studied the rate of uptake of ethylene in rats. They found that at equilibrium, which was rapidly attained, the rate of uptake followed Michaelis-Menten kinetics, where V_{max}, the maximum rate of uptake, was 0.24 $mg.kg^{-1}.h^{-1}$, and K_m, the atmospheric concentration of the substrate that causes uptake to proceed at half-maximal velocity, was 250 $mg.m^{-3}$ (220 ppm). Saturable uptake appeared to be associated with enzyme-mediated metabolism (Osterman-Golkar and Ehrenberg 1982).

Little information is available concerning the uptake and retention of ethylene by various tissues. Salanitre et al. (1965) calculated that the adipose tissue/blood partition coefficient in humans was 7.3; the half-time for ethylene uptake in adipose tissue was estimated to be between 70 and 400 min. Papper and Kitz (1963, cited by

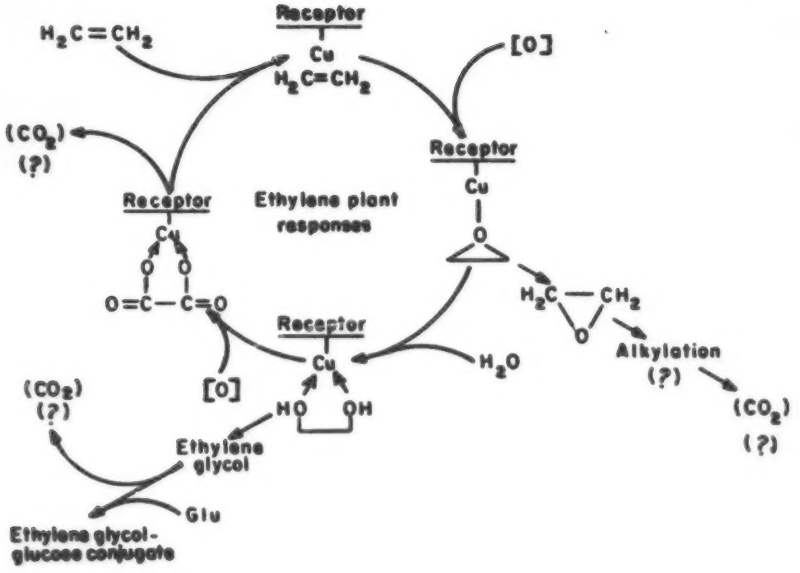


Figure 6-3. Tentative working hypothesis for initial events involved in ethylene metabolism and action (from Beyer 1981).

Miller 1969) concluded that absorption of ethylene was highest in lipoid tissues and lipoid cellular stroma. Guest et al. (1981) found that ethylene was not appreciably accumulated in rats exposed to ^{14}C-ethylene at 11 500 $mg.m^{-3}$ (10 000 ppm) for 5 h, although detectable ^{14}C levels were found in the blood, gastrointestinal tract, kidneys and liver 36 h after exposure.

Salanitre et al. (1965) measured the pulmonary uptake of ~11 500 $mg.m^{-3}$ (10 000 ppm) ethylene in humans; the expired concentration of the gas reached 95% of the equilibrium concentration in 5 min, 97% in 10 min, and 99% in 20 min. This suggests that the time course of the total body uptake of ethylene from the lungs is very short (Guest et al. 1981); the rapid equilibration probably reflects the low aqueous solubility of ethylene (blood:gas partition coefficient of 0.14; Anon. 1968) (Salanitre et al. 1965) and its slow rate of metabolism. Filser and Bolt (1983) found that the metabolic elimination of ethylene in rats saturated at ethylene concentrations above 1150 $mg.m^{-3}$ (1000 ppm).

Although the main route of elimination of inhaled ethylene appears to be exhalation, urinary excretion also plays a role (Ehrenberg et al. 1977). Rats have been observed to convert ^{14}C-ethylene to $^{14}CO_2$ and labelled urinary products (Van Dyke and Chenoweth 1965; Guest et al. 1981). Exhalation of labelled CO_2 was the explanation offered by Ehrenberg et al. (1977) for the plateau in radioactivity levels in the ^{14}C-ethylene exposure chamber of mice. Miller (1969) cited reports of several investigators which concluded that ethylene was eliminated unchanged, principally by the lungs, slightly in urine and gastrointestinal gases, and at a rate of 0.0019 $mg.cm^{-2}.h^{-1}$ (68 $nmol.cm^{-2}.h^{-1}$) by skin, most elimination being completed within a few minutes of administration.

The results of early investigations implied that ethylene was not metabolized by humans and other animals. However, many olefins are known to undergo epoxidation via a mixed function oxidase, often as an intermediate step in their transformation to more water-soluble compounds (glycols), and recent work seems to suggest that ethylene may be converted to ethylene oxide (see also Sections 6.2.1, 6.2.2.2) in some organisms.

Filser and Bolt (1983) reported that rats exposed to ethylene concentrations above 1150 $mg.m^{-3}$ (1000 ppm), for example, exhaled ethylene oxide, resulting in external concentrations of the epoxide of 1 $mg.m^{-3}$ (0.6 ppm) after 45 min. A subsequent decline in concentration of the epoxide occurred, so that a final concentration of 0.5 $mg.m^{-3}$ (0.3 ppm) was reached after several hours (Filser and Bolt 1984b). The authors suggested that exhalation of the reactive intermediate implies a distribution among various organs and tissues of the rat. Low levels of epoxide exhaled by unexposed rats were explained by endogenous ethylene production (Filser and Bolt 1983). Using the levels of ethylene oxide exhaled by rats, Filser and Bolt (1984b) calculated that about 70% of

the epoxide produced endogenously from inhaled ethylene is not systemically available. The authors suggested that this was indicative of detoxification of the majority of the compound close to its site of formation, probably in the liver.

Ehrenberg et al. (1977) noted the occurrence of labelled hydroxyethylcysteine in the urine of mice exposed to ^{14}C-labelled ethylene; since ethylene oxide is known to alkylate cysteine as well as histidine residues in hemoglobin, this suggested that at least some of the ethylene was metabolized to ethylene oxide. The authors proposed that the reaction occurred via a detoxification system which involved a mixed function oxygenase and epoxide glutathione transferase. Ehrenberg et al. (1977) calculated the amount of epoxide formed from the degree of alkylation of cysteine and histidine in hemoglobin; about 5.6% of the total hemoglobin radioactivity was found as the alkylation product hydroxyethylcysteine, and about 6.6% as hydroxyethylhistidine. Hemoglobin and liver proteins contained approximately the same amounts of labelled hydroxyethylcysteine, although metabolic incorporation into liver proteins was an order of magnitude greater than into hemoglobin. This suggests that the ethylene oxide is rapidly distributed in the body so that all organs receive the same tissue dose.

Support for the metabolic pathway for ethylene suggested by Ehrenberg et al. (1977) comes from the fact that the ratio between alkylated cysteine and alkylated histidine is the same as that found in mice after they had been exposed to ethylene oxide (Osterman-Golkar 1975, cited by Osterman-Golkar and Ehrenberg 1982). In addition, Segerbäck (1983) demonstrated that the ratio between the degree of alkylation of deoxyribonucleic acid (DNA) (measured as N-7-hydroxyethylation of guanine in DNA) and that of hemoglobin obtained after exposure of mice to ethylene and ethylene oxide was approximately the same, supporting the conclusion that ethylene oxide is a reactive intermediate formed in vivo from ethylene. His experiments showed that at low levels of ethylene, ~8% of the amount inhaled is metabolized to ethylene oxide; V_{max} (maximal rate of metabolism) was estimated to correspond to 7 mg.m^{-3} (4 ppm) ethylene oxide in air.

Schmiedel et al. (1983) studied the rat liver microsomal formation of ethylene oxide from ethylene in vitro, and found that the reaction proceeded in the presence of a regenerating system for NADPH (reduced nicotinamide adenine dinucleotide phosphate) with half-maximal velocity at a partial pressure of ethylene of 2.24 kPa. Because pretreatment with phenobarbital, which induces cytochrome P-450, or addition of metyrapone, which inhibits the phenobarbital-induced form of cytochrome P-450, had little effect on the transformation, the authors suggested that a different species of cytochrome P-450 is responsible for the conversion of ethylene to ethylene oxide.

If ethylene is in fact metabolized to ethylene oxide in humans and other animals, as it is in plants and microorganisms and as the above experiments seem to suggest, it is probable that the epoxide is further metabolized as it is in plants (see Section 6.2.2.2) and microorganisms (see Section 6.2.1). The short *in vivo* half-life of ethylene, about 10 min in mice and rats, cannot be explained by chemical reactivity (Ehrenberg *et al.* 1974; Osterman-Golkar *et al.* 1976, 1983); in water, the half-life of ethylene oxide is about 80 h at 37°C (Ehrenberg 1985). Martis *et al.* (1982), studying the pharmacokinetics of ethylene oxide administered intravenously in the dog, found that the epoxide cleared rapidly from the plasma ($\tau_{1/2}$ = 33 min) and was soon converted to ethylene glycol (mean plasma $\tau_{1/2}$ = 220 min), which can be further metabolized to glyoxylic acid, glycolaldehyde, oxalic acid and CO_2 (Gessner *et al.* 1961; Clay and Murphy 1977). Between 7 and 24% of the ethylene oxide was eliminated in the urine as the glycol within 24 h. None of the metabolites of ethylene glycol mentioned above, with the exception of CO_2, has thus far been reported in animals exposed to ethylene.

6.2.3.2 Ethylene Exposure Following Pretreatment

Several investigations of the uptake, retention, and elimination of ethylene by rats pretreated with chemicals that induce the hepatic mixed function oxidase system support the contention that ethylene is metabolized by monooxygenases. However, it should be remembered that quantitative and qualitative changes in metabolism (i.e. synergism or antagonism) could occur after these pretreatments.

Guest *et al.* (1981) exposed rats pretreated with Aroclor 1254, a polychlorinated biphenyl (PCB) mixture, to 11 500 $mg.m^{-3}$ (10 000 ppm) ^{14}C-ethylene for 5 h, and found that Aroclor pretreatment affected both elimination and tissue retention. The amount of ^{14}C-ethylene expired was not affected, but a 10-fold increase in expired $^{14}CO_2$ and a quadrupled urinary excretion of radioactivity were observed. Aroclor pretreatment also led to increases in the concentrations of radioactivity in the blood, gastrointestinal tract, kidneys and liver by factors up to 25-fold, and residual radioactivity in the brain, fat, heart, lungs, muscle, spleen and testes. The authors concluded that ethylene metabolism by a mixed function oxidase system was stimulated by Aroclor pretreatment, resulting in increased tissue and excreta concentrations of radioactivity associated with less volatile metabolites of ethylene rather than with the parent compound.

Filser and Bolt (1983) found that the maximal elimination rate (V_{max}) (8.5±1.0 $\mu mol.h^{-1}.kg^{-1}$) of ethylene was enhanced by Aroclor 1254 (13.7±2.6 $\mu mol.h^{-1}.kg^{-1}$) and the process was completely inhibited by diethyldithiocarbamate (dithiocarb) pretreatment. The authors concluded

that these results were consistent with the transformation of ethylene by monooxygenase(s) to ethylene oxide. Rats pretreated with dithiocarb and then exposed to ethylene exhaled much smaller amounts of ethylene oxide than did rats exposed to ethylene alone; this confirms the metabolic formation of ethylene oxide from ethylene by monooxygenases (Filser and Bolt 1983).

Several other experiments have been performed which imply that ethylene oxide is an intermediate in the hepatic metabolism of ethylene in PCB-pretreated rats (Conolly and Jaeger 1977; Conolly et al. 1977, 1978). However, since these studies examined the hepatotoxicity of ethylene and its metabolites, they will be discussed in detail in Section 7.3.

7.0 EFFECTS

7.1 SOIL MICROORGANISMS

Since ethylene is a volatile compound, it may have an effect on organisms spatially removed from its producers in the soil. Its role as a volatile inhibitor of fungal growth has been suggested by Smith (1973), who observed an inhibition of growth of Sclerotium rolfsii and Helminthosporium sativum propagules in soil when exposed to an air-stream containing ethylene and water-saturated air; propagules of test fungi grew only when exposed to ethylene-free air. This phenomenon, termed fungistasis, refers to the widespread inability of fungal propagules to germinate in soil under apparently favorable conditions unless supplied with exogenous nutrients (for reviews, see Dobbs et al. 1960; Jackson 1965; Smith 1973).

Since the first suggestion of ethylene's involvement in fungistasis, there has been little to substantiate that ethylene is a widespread fungistatic agent. Although Lynch (1975) confirmed that sclerotia of S. rolfsii were sensitive to ethylene, he showed that other fungi showed extensive hyphal growth in the presence of high levels of ethylene. Cornforth (1975) also noted healthy colonies of Botrytis cinerea, Penicillium, Trichoderma, and Mucor spp. growing on the soil surface in an ethylene atmosphere. Ethylene did not appear to affect the aerobic soil respiration. Therefore, the fungistatic effect of ethylene seems to be limited to a small number of species.

In conjunction with the hypothesis that the main producers of ethylene in soil are anaerobic, spore-forming bacteria, Smith (1976) postulated an ethylene cycle in soil. According to this scheme, the ethylene produced in anaerobic microsites diffuses through the soil and inactivates aerobic microorganisms and other less sensitive microbes in the vicinity of nutrients. The activity of the aerobes diminishes, thereby reducing the O_2 demand, which in turn allows O_2 to diffuse to the anaerobic microsites, inhibiting the activity of anaerobes. This permits resumption not only of aerobic growth, reducing O_2 in the microsite, but also of the ethylene-oxygen cycle. However, there has been little subsequent work by other scientists to support this theory since ethylene has not been shown to have an adverse effect on aerobic microbes at levels expected in the soil (Lynch 1975; Cornforth 1975; Smith 1978).

7.2 PLANTS

7.2.1 INTRODUCTION

Of all the air pollutants, or potential air pollutants, ethylene is remarkable in that it also acts as a plant hormone. Like

other plant hormones, it is synthesized by plants and can have profound effects on a wide range of aspects of growth and development. The description "hormone" is to some extent a misnomer. The word "hormone" is derived from the animal physiology literature, and plant hormones differ from their animal counterparts in at least five important respects: (i) plant hormones have different chemical structures and lower molecular weights; (ii) plant hormones show a relative lack of specificity and have a large overlapping range of effects; (iii) the mode of action of plant hormones appears to be different from that of animal hormones; (iv) the transport of plant hormones does not seem to be precisely controlled; and (v) the site of action of plant hormones may or may not be distant from the locus of biosynthesis, whereas for animal hormones only the former is true.

Although ethylene behaves in many respects like other plant hormones, it differs from the other four major groups (abscisic acid (AB), auxin, cytokinins and gibberellins (GA)) in that it is the only gaseous hormone. Thus ethylene can move not only through the liquid phase of a plant, but also through the internal air spaces and even from one plant to another (Denny and Miller 1935).

Of necessity this section is only a short review of the effects of ethylene; a large body of literature exists on the subject. For instance, Abeles (1973) has well over 1000 references on ethylene and Lieberman's short review (1979) lists 160 papers published between 1973 and 1978. For more in-depth information the reader is directed to the following reviews: Abeles (1973) - fairly exhaustive coverage; Abeles (1972) and Burg (1973) - general coverage but less exhaustive; Lieberman (1979) - biosynthesis and action; Osborne (1977) and Eisinger (1983) - cell growth and ethylene; also Skoog (1980), Wareing (1982), and MacMillan (1980).

7.2.2 FIELD OBSERVATIONS

Damage to shade trees by leaking illuminating gas was first reported over a hundred years ago. Although other gases in the illuminating gas could have caused the damage, the most important constituent was probably ethylene (e.g. Crocker 1913, cited by Abeles 1973).

In addition to damage from illuminating gas, ethylene from such sources as industries, greenhouses and automobiles has been observed to adversely affect fruit, flowers and nursery stock. For example, cotton grown in the vicinity of a polyethylene plant in Texas exhibited symptoms similar to those shown by cotton exposed to ethylene such as induced loss of leaves and leaf chlorosis (Hall et al. 1957). Premature abscission of mandarin fruit in the vicinity of a gas works has also been reported (Fukuchi and Yamamoto 1969, cited by Abeles 1982). A recent report on field studies in the Sarnia area of Ontario

demonstrated slight adverse effects on ethylene-sensitive bioindicator plants such as petunia and etiolated peas (Harper and Jones 1984). There are few, if any, additional reports in the literature of ethylene damage to vegetation in the vicinity of industries using or producing ethylene.

In contrast, there are far more reports of damage to crops in greenhouses from ethylene. During controlled atmosphere storage of fruit it is important to keep the ethylene level below threshold levels for activity, to effectively delay ripening (Dilley 1982). It is also commercially important to control the levels of ethylene during the storage of nursery stock (Curtis and Rodney 1952), flowers (e.g. Fischer 1950; Hasek et al. 1969; Barden and Hanan 1972; Hanan 1973b), and many other horticultural and agronomic plants (Heck and Pires 1962). However, in greenhouses, the burning of fossil fuels for heating and the provision of carbon dioxide enrichment of the atmosphere can produce ethylene that will damage the greenhouse crops (Hanan 1973a; Ashenden et al. 1977). Proximity to roads and major urban centers (Abeles 1982) and the production of ethylene from crops within the greenhouse itself (Eaves and Forsyth 1959) will have the same effect. In fact, the levels of ethylene produced by plants in a greenhouse can become sufficiently elevated to cause damage to plants near the greenhouse (Eaves and Forsyth 1969).

Finally, there is little information on the effects of automobile exhaust on vegetation in the vicinity of roads and highways. Although Flückiger et al. (1979) observed that the peroxidase activity of birch trees was higher in plants located on a highway median than in trees located at a distance from the highway, the traffic wind was found to place significant mechanical stress on the trees which induced a 340% increase in ethylene production.

7.2.3 PHYSIOLOGICAL AND BIOCHEMICAL EFFECTS

An exogenous supply of ethylene has a surprisingly large number of different effects on plants. In some of the earlier work it was sometimes assumed that because exogenously applied ethylene had a particular effect, the endogenous (internal) ethylene controlled this parameter of growth and development. That conclusion is, of course, not necessarily correct, and it is often rather difficult to prove that such parameters are indeed normally regulated by this hormone. In this regard, the use of antagonists of ethylene biosynthesis (see Figure 7-1) and action can be very useful (see Section 6.2.2.3). A range of these compounds, a few of which are shown in Table 7-1, is available. One must be careful that when these chemicals are applied to a plant, the effects observed are not due to toxic side effects unrelated to presumed anti-ethylene effects of the added substance. If the influence of the antagonist can be largely overcome by the readdition of ethylene, an ethylene-releasing compound, or ethylene precursors, it is an indication that the antagonist might not be exhibiting toxic side effects.

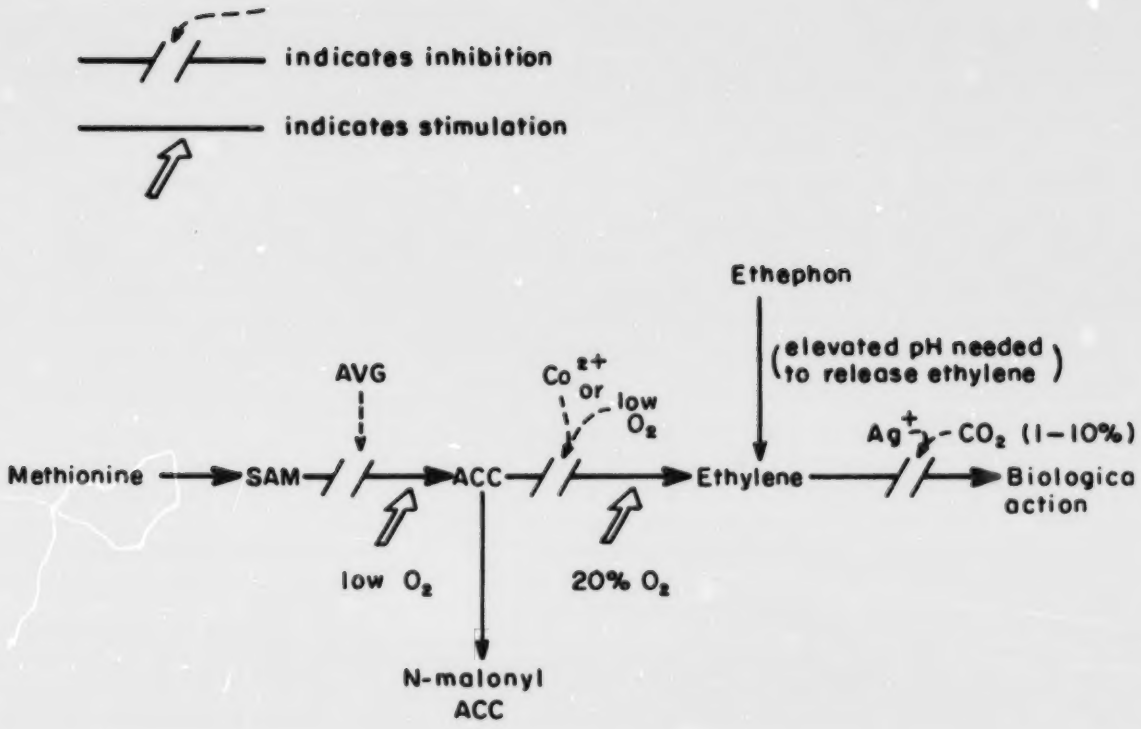


Figure 7-1. Site of action of some inhibitors of ethylene biosynthesis.

Table 7-1. Antagonists of ethylene biosynthesis and action.

Inhibitor	Reference
Ethylene biosynthesis	
Aminoethoxyvinyl glycine (AVG)	Pruess et al. (1974)
1-Canaline	Murr and Yang (1975)
Benzylisothiocyanate (BITC)	Patil and Tang (1974)
Diiodohydroxybenzoic acid (DIHB)	Robert et al. (1975)
N-benzynoxycarbonyl-L-α-aminocarboxy propionic acid; α-aminoxyacetic acid	Amrhein and Wenker (1979)
Rhizobitoxin	Owens et al. (1971)
Cobalt ions (Co^{2+})	Lau and Yang (1976)
Ethylene action	
Silver ions (Ag^{+})	Beyer (1976)
Carbon dioxide (CO_2) (1-10% can block ethylene action)	Burg and Burg (1967); Abeles (1973) (Table 9-3)
Carbon dioxide (>0.11% act antagonistically to ethylene in regulation of leaf axil angle)	Dhawan et al. (1981)

These antagonists (CO_2, Ag^+) could also be used in situations in which an exogenous supply of ethylene is suspected of affecting plant growth and development. Table 7-2 lists a series of effects that can be produced by ethylene supplied either as a gas or through the use of an agent that releases the gas inside the plant. Although not exhaustive, the list should give some idea of the scope of effects, which range from effects on growth and development to very specific influences upon biochemistry. The references were not always chosen, as is often the case, because they represent the first report of a particular finding, but rather because they very clearly showed the effect or contained useful discussion on the topic.

7.2.4 MODE OF ACTION

As Table 7-2 clearly shows, ethylene has an amazingly wide variety of effects. These include influences on dormancy (pollen, apical dominance, seeds and underground storage organs), rate and orientation of cell expansion (leaves, stems, hypocotyls, petioles, pollen, tubes and roots), rate of aging (leaves, flowers and fruits), osmotic swelling of motor cells, development and differentiation (flowering, cell division, rooting, xylogenesis and shoot initiation), events that can only be seen under the microscope, and many specific biochemical processes. Although ethylene is often considered merely an inhibitor of elongation, this is not a good assessment of its overall activities. For instance, ethylene redirects rather than inhibits stem growth, in that cells become fatter instead of longer. In addition, as will be pointed out, many processes can be stimulated by ethylene.

It is not the intention of this report to enter into a detailed analysis of the theories concerning the modes of action of ethylene. For this the reader is directed to the reviews of Lieberman (1979), Osborne (1982), Kende et al. (1982) and Eisinger (1983). There are, however, some questions that can be clarified and summarized:

1. Can the opposite effects of ethylene on certain processes (e.g. stem elongation of different species) be adequately explained?

2. Can any generalizations be made about dose-response-time relationships?

3. Can any overall conclusions be reached regarding a common mode of action of ethylene over the very wide range of responses?

These questions will now be examined.

7.2.4.1 Contradictory Effects of Ethylene

Although ethylene has been reported to have both stimulatory and inhibitory effects on particular processes (anthocyanin synthesis,

Table 7-2. Physiological and biochemical effects of ethylene on plants[a] .

Physiological/biochemical process	Effect
Dormancy and Growth	
Seed dormancy	Et + germination (breaks dormancy), 0.01 $mg.m^{-3}$ in peanut (Ketring and Morgan 1969), clover (Esashi and Leopold 1969), lettuce (Abeles and Lonski 1969) and striga (Egley and Dale 1970), I : CO_2, temperature and light
Bud dormancy	(see also "Apical dominance") Et + bud growth (breaks dormancy) (Hall et al. 1957) [F], I : season, photoperiod and depth of dormancy
Sprouting of tubers, bulbs, and corms	Et + in potato tubers (Vacha and Harvey 1927), I : CO_2 (Burton 1952). Et + in gladiolus corms (Vacha and Harvey 1927) and in narcissus bulbs (Haber 1926, cited by Abeles 1973)
Pollen dormancy	Et + peach pollen germination, 0.01 $mg.m^{-3}$ (Buchanan and Briggs 1969) [F]
Stem elongation	Et - in meso- and xerophytes, 0.01 $mg.m^{-3}$ [M] (Knight et al. 1910), I : CO_2 (Burg and Burg 1967), light (Burg and Burg 1968). Response seen within a few minutes. Et + in hydrophytes 0.5 $mg.m^{-3}$ (Ku et al. 1970), I : auxins (Imaseki and Pjon 1970)
Stem hypertrophy and widening	Et + stem width in dicots, 0.01 $mg.m^{-3}$ (Knight et al. 1910) [M]. Et + mesocotyl width (Camp and Wickliff 1981) in light
Epicotyl/hypocotyl hook opening	Et -, 0.01 $mg.m^{-3}$ (Goeschl et al. 1967; Kang et al. 1967). Effect seen within 2 h. I : light, phytochrome, CO_2
Pollen tube elongation	Et +, 0.01 $mg.m^{-3}$ (Buchanan and Briggs 1969) [F]
Leaf growth	Et - in dicotyledons, 0.01 $mg.m^{-3}$ (Burg and Burg 1968; Abeles and Heggestad (p. 120 in Abeles 1973)) and in monocotyledons (Van Andel and Verkerke 1978, used Ethrel)

Table 7-2. (Cont'd)

Physiological/biochemical process	Effect
Dormancy and Growth (Cont'd)	
Leaf curling and rolling	Et + (Hitchcock et al. 1932)
Petiole epinasty/hyponasty	Et - or 0 epinasty in dicotyledons, depending upon species, 0.01 $mg.m^{-3}$ (Crocker et al. 1932), I : CO_2 (Dhawan et al. 1981; Bassi and Spencer 1982) [M], age of tissue. Effects observed within 1 h. Et + hyponasty in Arizona cypress (Blake et al. 1980) [F]
Fruit growth	Et + or - in fig fruits depending upon developmental stage (Maxie and Crane 1968) [F]
Root elongation (main and laterals)	Et - in pear, 1 $mg.m^{-3}$ (Chadwick and Burg 1970), possible I : auxins and geotropism. Et + in rice, 0.01-0.1 $mg.m^{-3}$ (Smith and Robertson 1971; Konings and Jackson 1979). Et - fern rhizoids, 0.01 $mg.m^{-3}$ (Miller et al. 1970)
Survival of tree seedlings after cold storage	Removal of ethylene during cold storage of Loblolly pine seedlings increases field survival and root growth potential (Barnett 1983)
Gerontology	
Leaf senescence (yellowing)	Et + (accelerates), 0.1 $mg.m^{-3}$ (Osborne 1968; Hall 1977), I : photoperiod, temperature, other hormones, light [M]
Leaf abscission	Et +, 0.01-1 $mg.m^{-3}$, depending on species (Morgan and Gausman 1966; Beyer and Morgan 1971), I : auxin and many environmental factors (see also "Drought")
Pollination and corolla abscission	Et and pollination + corolla abscission in Digitalus (Stead and Moore 1983) [F]
Flower senescence (fading)	Et +, effects can be seen as low as 0.002 $mg.m^{-3}$, depending upon species (Uota 1969; Kende and Hanson 1976) [M] (see also "Membrane senescence")

Table 7-2. (Cont'd)

Physiological/biochemical process	Effect
Gerontology (Cont'd)	
Fruit ripening	Et + in many species, 0.1 $mg.m^{-3}$ (reviews: Burg and Burg 1962; Hulme 1970/1971; Dilley 1982) (see also "Respiration"). May be related to drought-stimulated Et production in some species (Adato and Gazit 1974)
Swelling of Motor Cells	
Stomates/guard cells	Most reports indicate 0 effect (Pallaghy and Raschke 1972; El-Beltagy and Hall 1974), but Ethrel can influence closing/opening (Browning 1974; Vitagliano 1975) and in peanut Et closes abaxial stomates (Pallas and Kays 1982), may be I with CO_2, and other factors affecting stomatal movement
Pulvini (leaf movement)	Et -, 0.1 $mg.m^{-3}$ (Morgan and Baur 1970) [F]
Development	
Leaf, number of stomates	Et + ratio of stomates/epidermal cells adaxial surface. Et - ratio of stomates/epidermal cells on abaxial surface (Funke et al. 1938, cited by Abeles 1973)
Male sterility	Ethrel + in wheat (Rowell and Miller 1971) [F]
Flowering	Et + rate in some species, especially Bromeliaceae, often 6-12 h of 10-115 $mg.m^{-3}$ needed (Traub et al. 1940), I : auxins. Et - flower initiation in some short day plants (e.g. in Xanthium), 1 $mg.m^{-3}$ during inductive night (Abeles 1967). Et + femaleness, continuous 0.1 $mg.m^{-3}$ in cucumber, especially in Canabinaceae, Cucurbitaceae, Eurphorbiaceae. Ethrel commonly used (McMurray and Miller 1969; George 1971; Abeles 1973, p. 140) [M]. Et + male sterility using Ethrel (Rowell and Miller 1971)
Apical dominance and tillering in monocotyledons	Et + in oats, 5 $mg.m^{-3}$ (Harrison and Kaufman 1982), I : cytokinins [F]. Et + internode growth in vegetative tillers (Van Andel and Verkerke 1978)

Table 7-2. (Cont'd)

Physiological/biochemical process	Effect
Development (Cont'd)	
Lateral bud outgrowth in dicots	Et - in peas, 3 $mg.m^{-3}$ (Blake et al. 1983), I : auxins [F]
Initiation of adventitious roots	Et + (Zimmerman and Hitchcock 1933). Et - (Coleman et al. 1980). Et + and - depending upon developmental stage (Fabijan et al. 1981), I : auxins (Mullins 1972; Wample and Reid 1979), light and water stress (Rajagopal and Andersen 1980)
Number of root hairs	Et + (Zimmerman and Hitchcock 1933) [F]
Differentiation, Callus, Cultures and Explants	
Shoot initiation	Et + (Bouriquet 1972; Lefebvre 1972; Cornejo-Martin et al. 1979) [F]. Et + or - depending upon stage of development (Huxter et al. 1981) [F]
Differentiation in xylem	Et + xylogenesis (Miller and Roberts 1982) [F]
Cell division	Et - (Burg et al. 1971; Van Andel and Verkerke 1978) [F] (see also "DNA synthesis")
Hypertrophy of lenticels (intumescences on woody stems)	Et +, 0.01 $mg.m^{-3}$ (Wallace 1928) [F]
Biochemistry	
Respiration	Et + respiration rate in many fruits (review: Hulme 1970/1971) and tubers (Reid and Pratt 1972; Theologis and Laties 1982)
Mitochondrial swelling	Et +, 10 $mg.m^{-3}$ (Lyons and Pratt 1964; Olson and Spencer 1968a, b; Olson et al. 1968; Ku and Leopold 1970) [F] (see also Section 7.3)
Photosynthesis	Et may - net photosynthesis indirectly via stomatal closure [F] (Pallas and Kays 1982) and reduced leaf area (Abeles and Heggestad in Abeles 1973) (see also "Chlorophyll degradation" and "Loss of chloroplast structure")

Table 7-2. (Cont'd)

Physiological/biochemical process	Effect
Biochemistry (Cont'd)	
Carotenoid synthesis	Et -, 0.01 $mg.m^{-3}$ (Kang and Burg 1972) [F]
Chlorophyll degradation	Et +, 0.1 $mg.m^{-3}$ (Nakagaki et al. 1970). Et + chlorophyllase activity (Looney and Patterson 1967)
Anthocyanin synthesis	Et +, 0.1 $mg.m^{-3}$ (Craker 1971) [F]. Et -, 0.01 $mg.m^{-3}$ (Craker et al. 1971) [F]
α-Amylase	Et + synthesis and release in barley aleurone cells at early stages of germination (Ho et al. 1982; Eastwell and Spencer 1982a, b, c), I : gibberellins
Cellulase synthesis and secretion	Et + (Horton and Osborne 1967)
Catalase activity	Et + (Mattoo and Modi 1969), Ethrel + (Henry and Jordan 1977), I : IAA, GA
Phenylalanine ammonium-lyase (PAL) and enzymes involved in lignification	Et +, between 0.001 and 1 $mg.m^{-3}$ (Riov et al. 1969; Morgan and Fowler 1972; Chalutz 1973; Rhodes et al. 1976; Hyodo et al. 1978; Rhodes and Wooltorton 1978). Et - (Morgan and Fowler 1972)
Peroxidase	Et +, 1 $mg.m^{-3}$ (Imaseki 1970; Van Loon 1977) [M]. Ethrel + (Henry and Jordan 1977)
Chitinase	Et +, 10 $mg.m^{-3}$ (Boller et al. 1983)
Cell wall components (carbohydrate polymers)	Et (1-60C $mg.m^{-3}$) can - or + level of cell wall precursors (Terry et al. 1981), I : auxins (reviews: Osborne 1982; Eisinger 1983)
β-1,3-Glucanase	Et + (Abeles and Forrence 1970). Et - (Wong and Maclachlan 1980)
Polygalacturonase synthesis	Et + in tomato fruit (Grierson and Tucker 1983)

Table 7-2. (Cont'd)

Physiological/biochemical process	Effect
Biochemistry (Cont'd)	
DNA synthesis	Et - DNA synthesis (Burg and Kang 1972; Sato et al. 1976). Et - (reduces levels) DNA polymerase (Apelbaum et al. 1974)
Protease activity	Et + in gibberellic acid - treated barley aleurone layers (Eastwell and Spencer 1982c)
Redistribution of ions	Et + release of Ca^{2+} from gibberellic acid - treated aleurone layers by 101%; release of Ca^{2+} was much more sensitive to ethylene than were changes in distribution of K^{+}, Mg^{2+} or phosphate (Eastwell and Spencer 1982b)
RNA and protein synthesis	Et small + in RNA synthesis, soybean hypocotyl segments (Holm et al. 1970), I : auxins. Et alters type of polypeptides in soybean hypocotyl segments (Zurfluh and Guilfoyle 1982). Et + loss of protein in senescent tissues (Abeles and Holm 1967)
Xylanase activity	Et + development by barley aleurone layers exposed to gibberellic acid (Eastwell and Spencer 1982b)
Mitochondrial ATPase	Et + (Malhotra and Spencer 1974) (see also Section 7.3)
Hydroxyproline-rich glycoproteins	Et + (Ridge and Osborne 1971; Esquerré-Tugayé et al. 1979) [F]
ACC synthase (an enzyme of the Et biosynthetic pathway)	Et +, 13.2 $mg.m^{-3}$ for 48 h in tomato fruit (Kende et al. 1982)
pH of latex cytoplasm	Ethrel + (Tupy 1980) [F]
Ion and sucrose efflux, membrane permeability	Et + ion and sucrose efflux in morning glory flower tissue (10 $\mu L.L^{-1}$ (ppm)) (Hanson and Kende 1975) [F]
Membrane senescence	Et + in cut flowers (Thompson et al. 1982)

Table 7-2. (Cont'd)

Physiological/biochemical process	Effect
Transport	
Auxin transport	Et − in land plants (Burg and Burg 1967; Beyer and Morgan 1971; Beyer 1975c). Et + in aquatic plants (Musgrave and Walters 1973)
Exudation	Et + guttation (Hall et al. 1957), exudation of sap and gummosis in trees (Buchanan and Briggs 1969) and latex flow in rubber trees. Ethrel rather than Et is commonly used (review: Leopold 1971)
Effects Seen at Microscopic Level	
Xylogenesis	Et + (Miller and Roberts 1982)
Microtubule orientation	Et alters (Steen and Chadwick 1981; Lang et al. 1982)
Cellulose microfibril orientation	Et alters (Lang et al. 1982; review: Eisinger 1983)
Loss of chloroplast structure (during senescence)	Et may + (Abeles 1973, p. 156)
Mitochondrial development	Et + (Makimoto and Asahi 1981) (see also "Mitochondrial swelling" under Biochemistry)
Cell division	Et − in leaves (Burg et al. 1971) and fern rhizoids (Miller et al. 1970) [F]

Note: 1 ppm ≈ 1.15 $mg.m^{-3}$ ethylene.

[α] Et, ethylene; +, promotes; 0, no response; −, inhibits; I, interaction with stated factor; [M], many confirmatory reports; [F], few confirmatory reports; 0.01 $mg.m^{-3}$ or 50 $mg.m^{-3}$ and so on, indicates normal lower limit of effects.

phenylalanine ammonium-lyase activity, level of cell-wall precursors, adventitious rooting, shoot initiation, flowering, root and stem elongation; see Table 7-2), this may not be quite as contradictory as it first appears for the following reasons:

(i) Application of the plant hormone auxin produces the biphasic dose-response curve shown in Figure 7-2, curve A. However, the whole curve can shift to the left (B), or to the right (C), depending upon such factors as type and age of the tissue under study. For instance, it has been known for many years that roots are more sensitive to auxin than are buds, which in turn are more sensitive than shoots (Leopold 1955; Thimann 1969). The response of roots, buds and shoots would then follow curves B, A and C, respectively.

Although such biphasic dose-response curves have not been commonly reported in ethylene studies, they do exist. Roots of rice exhibit a biphasic growth response to ethylene (Konings and Jackson 1979; Jackson 1982), and Reid and Watson (1984) have found similar effects in experiments in which plants have been constantly exposed to very low levels of ethylene. In those experiments the control treatment of "zero" ethylene was not simply untreated laboratory air but was air that had been scrubbed of ambient ethylene so that it contained less than 0.06 $\mu g.m^{-3}$ (0.05 ppb) ethylene.

Zobel (1973) worked with a tomato mutant which exhibited abnormalities of growth and development. These lesions could be overcome by the addition of very low (6 $\mu g.m^{-3}$; 5 ppb) levels of ethylene. Other studies with this mutant and with tracheid differentiation in lettuce pith also indicated that low levels of ethylene may promote various processes while higher concentrations are inhibitory (Zobel 1974; Zobel and Roberts 1978).

(ii) If, as suggested by Lieberman and Kunishi (1972), Zobel (1974), and Zobel and Roberts (1978), tiny quantities of endogenously produced ethylene are constantly required for the control of growth and development, it would be difficult to observe the promotive portion of the biphasic curve by additional applications of the gas. This presumptive part of the dose-response curve could be seen only through the use of mutants (Zobel 1973), antagonists of ethylene action or synthesis (Fabijan *et al.* 1981; Huxter *et al.* 1981), or perhaps by growing plants in lower than ambient ethylene concentrations under conditions of low pressure.

(iii) One difficulty in interpreting the results of many workers is that laboratory air is always contaminated with a low background concentration of ethylene. Even more unfortunately, the concentration of ethylene in air differs from location to

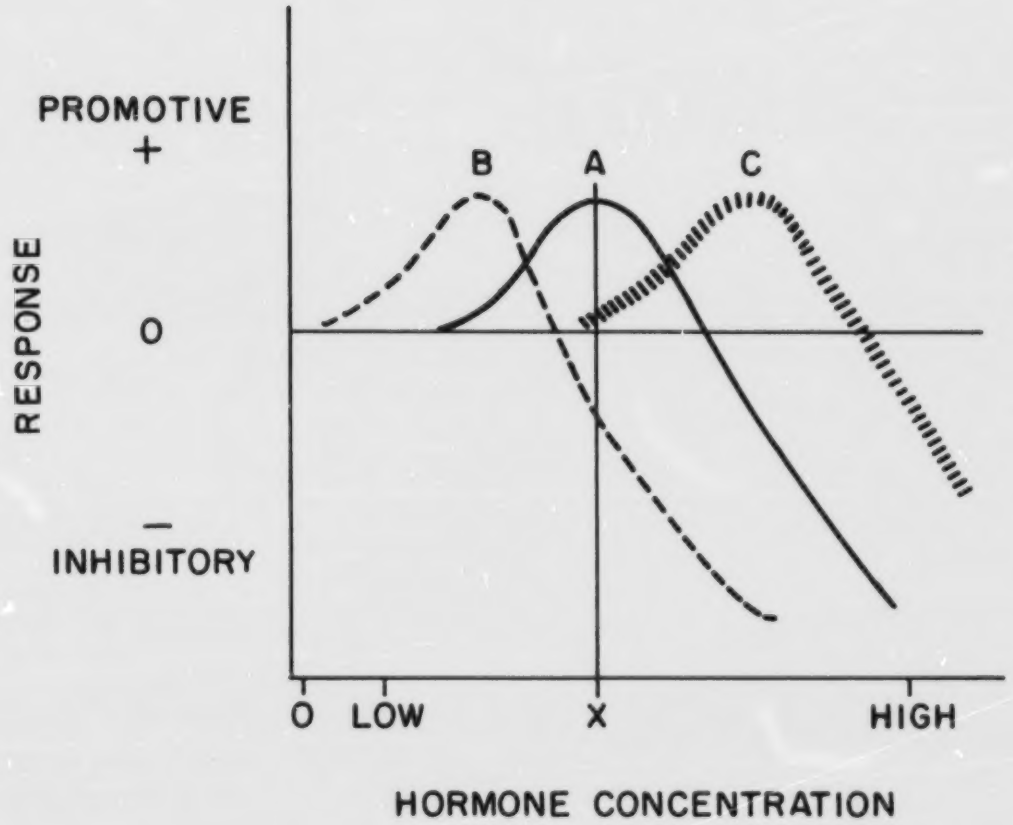


Figure 7-2. Biphasic dose-response curves for plant hormones. Concentration X of the hormone may have (i) promotive, (ii) no, or (iii) inhibitory effect depending upon the type of sensitivity curve. The type of curve may depend upon such factors as type or age of tissue.

location and also shows substantial diurnal variation (Abeles and Heggestad 1973; Harbourn and McCambley 1973; Guicherit 1975; Nassar and Goldbach 1979; Reid and Watson 1981 (see Tables 5-3 and 5-4 in Chapter 5)).

Consequently, unless the appropriate precautions are taken, one is never quite sure of the ethylene concentration of the experimental controls, and one might fail to observe effects of low levels of ethylene.

(iv) Another problem in obtaining the correct interpretation of experimental results is that some cells may be responsive to ethylene only for a short period, after which they lose their ability to react (Osborne 1982). As well, in some tissues ethylene initially has an inhibitory effect on a particular aspect of growth and development, yet within hours (Fabijan et al. 1981) or days (Huxter et al. 1981) that same aspect is stimulated by the gaseous hormone. Trewavas (1982) has pointed to a number of systems in which tissues may pass through phases of altering sensitivity to plant hormones; also, as mentioned earlier, the ability of a particular level of ethylene to promote cell elongation in aquatic plants and yet to have the opposite effect on other species may merely indicate that the former are less sensitive to the gas (Table 7-3; also see comments regarding dose-response curve).

7.2.4.2 Dose-Response-Time Relationships

Although there are many data, albeit incomplete, on dose-response relationships, there is less work on dose-response-time relationships (Tables 7-4 and 7-5). In addition, a significant proportion of the published works on ethylene have used only one or two dose rates of ethylene in each experiment. Furthermore, especially in earlier work, rather high levels of the hormone have been used. Except in fruit tissue, the internal levels of ethylene are normally well below 1 $mg.m^{-3}$ (0.9 ppm) (Abeles 1973). Thus giving an exogenous supply greatly in excess of those levels may merely produce hard-to-interpret (unnatural) pharmacological effects.

Abeles (1973) has suggested that 0.01 $mg.m^{-3}$ (ppm) ethylene is needed for a threshold effect, and 0.1-0.6 $mg.m^{-3}$ (0.1-0.5 ppm) for a maximum effect. Many factors can act to modify these lower and upper limits:

(i) As discussed earlier, the low but ever present and variable levels of background ambient ethylene could interfere.

(ii) The length of exposure to the gas is frequently ignored and, especially for the threshold limit, would be an important parameter.

Table 7-3. Sensitivity of selected plants to ethylene (from Heck et al. 1970).

Sensitive	Intermediate	Resistant
Bean, Black Valentine (Phaseolus vulgaris)	Arbovitae (Thuja orientalis)	Beet (Beta vulgaris)
Carnation (Dianthus caryophyllus)	Azalea (Rhododendron sp.)	Cabbage (Brassica oleracea)
Cotton (Gossypium hirsutum)	Carrot (Daucus carota)	Clover (Trifolium sp.)
Cowpea (Vigna sinensis)	Gardenia (Gardenia radicans)	Endive (Cichorium endivia)
Cucumber (Cucumis sativus)	Holly, Japanese (Ilex crenata)	Grass, rye (Lolium multiflorum)
Marigold, African (Tagetes erecta)	Soybean (Glycine max)	Oats (Avena sativa)
Orchid (Cattleya sp.)	Squash (Cucurbita maxima)	Onion (Allium cepa)
Pea, cream (Pisum sativum)		Radish (Raphanus sativus)
Peach (Prunus persica)		Sorghum (Sorghum vulgare)
Philodendron (Philodendron cordatum)		
Privet (Ligustrum sp.)		
Rose (Rosa sp.)		
Sweet potato (Ipomoea batatas)		
Tomato (Lycopersicon esculentum)		

Table 7-4. Some reported dose-response-time relationships for ethylene and vegetation.

Exposure Concentration ($\mu g.m^{-3}$)	Exposure Time (h)	Effect	Reference
		Orchids	
345	1	slight injury	Darley et al. (1963)
58	6	slight injury	Darley et al. (1963)
46-115	8	typical to extensive injury	Davidson (1949)
2.3-23	24	slight to typical injury	Davidson (1949)
12	24	slight injury	Darley et al. (1963)
		Carnations	
115	6	closes flowers	Darley et al. (1963)
480	12	closes flowers	Crocker and Knight (1908)
230	24	closes flowers	Nichols (1968)
140	24	threshold for injury	Uota (1969)
35	48	threshold for injury	Uota (1969)
5,060, 13 800 and 54 000 $\mu g.m^{-3}.h$		open flower keeping life reduced (compared with controls) to 0% at 21.1, 10.0 and 1.7°C, respectively	Barden and Hanan (1972)
		Snapdragon	
580	1	petals drop	Darley et al. (1963)
		African Marigold	
20	48	plant epinasty	Crocker et al. (1935)
58	12	plant epinasty	Crocker et al. (1932)
		Pea	
12	12-60	8.5% mean inhibition of epicotyl elongation	Goeschl and Kays (1975)
23	"	22% mean inhibition	Goeschl and Kays (1975)
46	"	30% mean inhibition	Goeschl and Kays (1975)
92	"	44% mean inhibition	Goeschl and Kays (1975)
184	"	60% mean inhibition	Goeschl and Kays (1975)

Table 7-4. (Cont'd)

Exposure Concentration ($\mu g.m^{-3}$)	Exposure Time (h)	Effect	Reference
		Petunia	
136	6	44% increase in dead blooms 24 h after fumigation (19% in control with 17 $\mu g.m^{-3}$); bloom diameters decreased by 2.4% 24 h after fumigation (1.6% in control)	Harper and Jones (1984)
136	7-9	53% increase in dead blooms; bloom diameters decreased by 6.5%	Harper and Jones (1984)
		Tomato	
115	several	flower buds drop (also pepper)	Darley et al. (1963)
460	3	plant epinasty	Crocker et al. (1932)
115	48	plant epinasty	Crocker et al. (1932)
		Cotton	
46-3450	during growing season	100% loss in yield within 1 mile of polyethylene plant	Heck et al. (1961)
		Potato	
58	16	plant epinasty	Denny and Miller (1935)
29	48	plant epinasty	Crocker et al. (1935)
		Buckwheat	
58	12	plant epinasty (also chenopodium and sunflower)	Crocker et al. (1932)
		Apple	
12	48	intumescences on apple cuttings	Wallace (1927)
		Greenhouse crops	
58	8	significant losses	Abeles (1973)

Note: 1 ppb = 1.15 $\mu g.m^{-3}$ ethylene.

Table 7-5. Key criteria (selected from Table 7-4).

Exposure Concentration ($\mu g.m^{-3}$)	Exposure Time (h)	Effect	Reference
115	several	flower buds drop - tomato and pepper	Darley et al. (1963)
115	6	close flowers - carnation	Darley et al. (1963)
58	12	epinasty - buckwheat, sunflower and African marigold	Crocker et al. (1932)
58	16	epinasty - potato	Denny and Miller (1935)
29	48	epinasty - potato	Crocker et al. (1935)
20	48	epinasty - African marigold	Crocker et al. (1935)
12	48	intumescences on twigs - apple	Wallace (1927)
12	24	threshold level for effects on vegetation	Neljubow (1901); Abeles (1973); Abeles and Heggestad (1973)

Note: 1 ppb = 1.15 $\mu g.m^{-3}$ ethylene.

(iii) The age and type of tissue may modify the response (see Table 7-2). This is especially important in senescent tissues and in ripening fruit. In the latter a much higher threshold exists. Abeles (1973) argues that this could be due to the high rate of production of ethylene in fruits.

A question that has not been examined in detail is the possibility that a history of environmental stress (e.g. drought) might modify subsequent responses to exogenous ethylene. A number of reasons may be suggested for such an effect: (i) stress may alter the ability of the plant to respond; (ii) environmental stress often results in an elevated rate of endogenous ethylene production (see Table 7-6), and this might modify the effect even more than the ethylene being supplied from outside of the tissue; and (iii) stress alters the levels of other phytohormones which are known to modify ethylene action and rate of production.

As an extension of this, since so many environmental parameters (Table 7-6) can either alter the responses of the plant to added ethylene or influence internal ethylene production, a great deal of careful control must be exercised over the growth and treatment of experimental plants. For example, on a winter day CO_2 levels in an experimental greenhouse connected to a building housing large numbers of students at the University of Calgary were found to be in excess of 1800 $mg.m^{-3}$ (1600 ppm) (Guy 1984). This concentration of CO_2 might be enough to antagonize the effects of ethylene (in the range of 11-115 $\mu g.m^{-3}$ (10-100 ppb) ethylene) or even influence the rates of ethylene production (Dhawan et al. 1981).

It is interesting to speculate on the global problem of increasing atmospheric levels of CO_2. Will concentrations of CO_2 ever reach a level at which it would start to act as an inhibitor of ethylene action in plant tissues?

There appears to be little information about a possible effect of chronic or acute elevated ethylene levels on winter dormant forest trees. Barnett (1983) reported that lowering ethylene levels around cold stored Loblolly pine seedlings will increase the survival of the seedlings when they are eventually planted in the field.

7.2.4.3 Action at the Molecular Level

Much of the recent effort in ethylene research has fruitfully concentrated on the pathway of ethylene biosynthesis and the controls over that pathway. However, much less success has attended our efforts to discover ethylene's molecular mode of action. As Table 7-2 shows, ethylene acts on such a plethora of biochemical, growth and differentiation parameters that it is reasonable to argue that the gas must be able to control some fundamental and central process.

Table 7-6. Interactions with environmental and other variables.[a]

Variable	Effect
Injury in general	+ Et production (Wright 1978; Kimmerer and Kozlowski 1982)
Freezing	+ Et production (Elstner and Konze 1976)
Gravity	Exogenous Et interferes with response of stems and petioles to gravity (Crocker et al. 1932; Hall et al. 1957). Endogenous Et production increases during upward bending of horizontally oriented shoots (Bucher and Pilet 1981; Salisbury et al. 1982; Clifford et al. 1983)
Drought	+ Et production (McMichael et al. 1972; Wright 1977, 1980), I : other plant hormones especially abscisic acid and cytokinins
Flooding/waterlogging	+ Et production in shoots (aerated or nonflooded portions of plant) (Kawase 1974; review: Reid and Bradford 1984)
Anoxia (low O_2)	- Et production (blocks ACC to Et conversion) only in those tissues that are anoxic. Anoxia also + ACC synthesis (Bradford et al. 1982)
Application of synthetic auxin-like herbicides and auxins	+ Et production (Rubinstein and Abeles 1965; Imaseki et al. 1982) [M]
Wounding	+ Et production (Hanson and Kende 1976; Yang and Pratt 1978) [M], within 20-30 min (Jackson and Campbell 1976a)
Mechanical (touching, pressure, friction, wind)	+ Et production (Goeschl et al. 1966; Jaffee 1970; Jaffee and Biro 1977; Saltveit and Dilley 1978)
SO_2 (0.3 $\mu L.L^{-1}$ (ppm))	+ Et production (Kimmerer and Kozlowski 1982)
Gibberellins	Antagonize Et effects on cell elongation (Stewart et al. 1974)

Table 7-6. (Cont'd)

Variable	Effect
Cytokinins	+ Et production and enhance auxin-induced Et production (Fuchs and Lieberman 1968; Imaseki et al. 1975) [M]
O_3 ($0.05\ \mu L.L^{-1}.h^{-1}$)	+ Et production (Tingey et al. 1976; Kimmerer and Kozlowski 1982)
CO_2	Antagonizes effect of Et (Burg and Burg 1967) [M]. CO_2 (0.5%) in the light and Et production in intact sunflower (Dhawan et al. 1981)
Light	+ Et production (Meheriuk and Spencer 1964; Abeles and Lonski 1969; Robert et al. 1975). - Et production (Huxter et al. 1979, 1981; Gepstein and Thimann 1980)
Pathogens (fungal, viral, bacterial and insects)	+ Et production and also + phytoalexin production and other chemicals perhaps allowing for defence against further pathogen attack (Imaseki et al. 1968; and reviews: Hislop et al. 1973; Pegg 1976; Boller 1982)
Cu^{2+}, Fe^{3+}, Hg^{2+}	+ Et production (Abeles 1973)
X-rays, gamma irradiation and ultraviolet	+ Et production in fruit tissue (Abdel-Kader et al. 1968; Chalmers and Faragher 1977)
Abscisic acid	Complex interaction with Et especially obvious in stem elongation and senescent tissues (review: Lieberman 1979, pp. 470-471)

[α] Et, ethylene; +, promotes; -, inhibits; I, interaction with stated factor; [M], many confirmatory reports.

Studies on structure-function relationships (Burg and Burg 1967; Abeles and Gahagan 1968; Beyer 1972; Abeles et al. 1972; Abeles 1973, pp. 220-223) show that among those studied, ethylene itself is the most potent compound of this nature. One interpretation of this might be that ethylene itself and not some converted, more active, form is exerting its effects in the cell.

There are some nongaseous chemicals that appear to mimic the physiological effects of ethylene. Ethrel (2-chloroethylphosphonic acid) appears to act like ethylene because it readily liberates ethylene at a pH above 4.5 (Yang 1969). In the presence of light and free radicals, other compounds will produce ethylene (Yang 1974). Abeles (1973) suggested that ethylene chlorohydrin might produce its ethylene-like effects by a similar mechanism. Methionine, S-adenosylmethionine (SAM) and 1-aminocyclopropane-1-carboxylic acid (ACC) mimic ethylene for the simple reason that they are its precursors. Abeles (1973) lists a number of unsaturated and chemically related (to ethylene) gases. None of these is as potent as ethylene, and it seems that for maximal activity the molecule must be small and unsaturated, with the unsaturated bond beside a terminal carbon that must be electrophilic.

It is not yet clear how ethylene acts at the molecular level. A number of avenues have been investigated, such as:

(i) the binding of ethylene to a metal at the site of action (Beyer 1972);

(ii) the interaction of ethylene with membranes (Mehard et al. 1970);

(iii) the close relationship between ethylene metabolism and its action (Beyer 1975a, b, 1981; Beyer and Sundin 1978; Dodds et al. 1979);

(iv) the binding of ethylene to various receptor sites (Bengochea et al. 1980; Sisler 1980); and

(v) the characterization of the immediate (or primary) response to ethylene (Kende et al. 1982).

All of these approaches have been valuable, but as yet none has definitely explained how ethylene operates at the molecular level. For instance, the work of Mehard et al. (1970) with artificial membranes showed that while ethylene influenced certain physical properties of the model system, other specific hydrocarbons were more effective. On the other hand, ethylene might bind some specific membrane-bound protein which would modify membrane activities. Various workers have produced evidence suggesting that labelled ethylene can bind to membranes, some of which could involve protein binding sites (Sisler 1980; Bengochea et

al. 1980; Evans et al. 1982). Much more work is needed here because it is difficult to show conclusively that the presumptive ethylene binding site is indeed the true biologically active one.

Other intensive studies are being undertaken to determine how closely metabolism and action of ethylene are related (see Section 6.2.2.3) and to explain why the affinity for ethylene in some tissues (peas) is much lower than in others (Vicia) (see Hall et al. 1982a for a discussion of this last point).

Finally, although it may not represent the primary mode of action of ethylene, the effect of the gas on microtubule orientation is of great interest. The orientation of microtubules in cells is of vital importance in the orientation of new cell wall cellulose microfibrils (Pickett-Heaps 1967; Newcombe 1969), and this is a major regulator of the rates and direction of cell expansion (Frey-Wyssling 1976; Maclachlan 1977). Ethylene alters the orientation of microtubules (Lang et al. 1962; Steen and Chadwick 1981), and thus can strongly influence cell growth. Similar effects on microtubules might also partially explain how ethylene interferes with cell division (see Table 7-2), since the orientation, polymerization and depolymerization of microtubules of the meiotic and mitotic spindles control the rate of cell division and segregation of the chromosomes to daughter cells.

7.2.5 EFFECTS OF METABOLITES

As has been pointed out in Section 6.2.2, ethylene may be metabolized to ethylene oxide and possibly ethylene glycol in certain plant species. It is therefore possible that some of the effects previously attributed to ethylene are in fact the results of action of one or more of ethylene's metabolites.

Little work has been performed to confirm these speculations, although the mutagenic effectiveness and efficiency of ethylene oxide on rice have been examined (Jana and Roy 1975); reductions in growth, survival and germination have also been observed (Roy and Jana 1975). Much work remains to be done in this area.

Ethylene oxide is also known to act synergistically with ethylene to inhibit growth in peas, and to amplify other effects (Beyer 1981, 1984). This increase was not the result of stimulation of endogenous ethylene production, raising the possibility that ethylene oxide may be directly involved in ethylene action. Although the levels of ethylene oxide used in these studies exceed physiological concentrations (based on rates of ethylene metabolism), ethylene oxide formed in vivo may be more effective than when applied exogenously (Beyer 1984). Ethylene oxide has also been observed to act as an ethylene antagonist (Abeles 1973).

7.3 HUMANS AND OTHER ANIMALS

7.3.1 SIMPLE ETHYLENE EXPOSURE

Ethylene was used for many years as an anesthetic for humans and other animals (Luckhardt and Lewis 1923; Luckhardt and Carter 1923). The concentration required for anesthesia in humans averages about 82% ethylene (with 18% oxygen), and a 15-s exposure induces sleep (Abeles 1973). However, because of the explosion hazard associated with the use of ethylene, it was replaced in this application by other gases.

Several workers have investigated the possibility that ethylene causes effects other than anesthesia and asphyxia in animals; the results of some of their experiments seem to confirm this possibility. In cats, for example, ethylene applied intraarterially depressed ganglionic activity through its action on the transmission of nerve impulses in the upper neck sympathetic ganglion (Medvedev 1959). Friede and Ebert (1934) found that ethylene reduced the growth rate of normal animal tissues in vivo as well as in tissue culture. The same effect of ethylene was observed with certain tumors, and the gas altered the capacity of the latter for implantation in healthy animals. On the other hand, Luckhardt and Carter (1923) found that several different species of animals anesthetized with 75-90% ethylene in oxygen for 30- to 100-min periods exhibited no noticeable side effects, except for short-lived nausea. Harvey (1928) exposed guinea pigs to 1150 $mg.m^{-3}$ (1000 ppm) ethylene via inhalation for 4 weeks, during which time he observed no effects on weight gain, no lesions of any organs, and no evidence of congestion or irritation of the lungs and respiratory passages. Similarly, Hirschfelder and Ceder (1930) found no effect on the growth of rats or on the action of lipase, esterase, pepsin or trypsin in rats exposed continuously to 11 500, 1150 and 115 $mg.m^{-3}$ (10 000, 1000 and 100 ppm) ethylene; however, a definite increase in amylase action was observed.

A study performed by Industrial Bio-Test Laboratories, Inc. (IBT)[a] for the Chemical Industry Institute of Toxicology (1977) concluded that ethylene was nontoxic to rats exposed continuously to the compound at 11 500, 3400, 1150 and 340 $mg.m^{-3}$ (10 000, 3000, 1000, 300 ppm) for 90 d. Although significantly lower liver weights were noted in several groups of exposed rats, the researchers claimed these were unlikely to be related to treatment because of the lack of dose-response effect and the lack of hepatocellular changes that usually accompany liver damage. No evidence of chronic toxicity or oncogenicity was

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[a] The Associate Committee on Scientific Criteria for Environmental Quality cautions against the use of any IBT data that have not been checked for validity.

observed in chronic inhalation studies in rats exposed continuously to ethylene at 3400, 1150 and 340 $mg.m^{-3}$ (3000, 1000, 300 ppm) for 24 months (Chemical Industry Institute of Toxicology 1980). The results of this study, also performed partially at IBT, were audited by a third party quality assurance group (Tracor Jitco, Inc. 1982) which concluded that the only errors noted in the final report were minor in nature and did not affect the conclusions drawn from the data. The results were recently published in the open literature by Hamm et al. (1984).

At the subcellular level, several experiments have shown similar effects of ethylene in both animals and plants. For example, Lyons and Pratt (1964) found that ethylene slightly increased the swelling of mitochondria from rat liver or cauliflower, but only when the particles were suspended in a medium containing 0.125 M KCl as the osmotic stabilizer. Others (Olson and Spencer 1968a, b; Olson et al. 1968), with more sensitive techniques, observed ethylene effects when mannitol (or sucrose) was used as osmoticant; these effects were more pronounced in KCl. Ethylene (23-115 $mg.m^{-3}$ (20-100 ppm) in air) had similar effects on mitochondria from yeast, rat liver and bean cotyledons (Olson and Spencer 1968a, b, c; Olson et al. 1968). Ethylene treatment resulted in an increase in the rate of mitochondrial volume changes caused by adenosine diphosphate or adenosine triphosphate. However, it had no effect on spontaneous volume changes or on those mediated by phosphate or calcium ions. In addition, the gas increased the rate of adenosine triphosphate hydrolysis by the mitochondria. These observations, taken together, suggested that ethylene affects mitochondrial adenosine triphosphatase (ATPase). Inhibitor studies supported this suggestion. Computer simulation of a model system indicated that the increase in adenosine diphosphate concentration that occurs when mitochondria are treated with ethylene would be sufficient to cause a major increase in mitochondrial, and hence cellular, respiration (Olsen et al. 1968). (Many reports record simultaneous increases in respiration and ethylene production by a number of tissues.) Further evidence that ethylene affects the activity of mitochondrial ATPases comes from the work of Malhotra and Spencer (1974) on isolated "membrane-bound" and "highly purified" ATPases from pea cotyledon mitochondria. Ethylene at concentrations of 2.5-2000 $\mu L.L^{-1}$ (ppm) was shown to stimulate the activity of the isolated ATPases. The effect was dependent on the presence of certain levels of carbon dioxide, and was augmented by phospholipids.

There is some evidence that in plants, under certain conditions, ethylene can enhance the activity of the alternative respiratory pathway ("cyanide-resistant respiration") (e.g. Rychter et al. 1979; Day et al. 1980; Lance 1981). The small amount of evidence currently available suggests that in animals this pathway is of limited occurrence (Henry and Nyns 1975). It does operate in a variety of microorganisms, under particular growth conditions.

Ethylene inhibits the rate of oxidation of the plant growth substance, indole-3-acetic acid, by peroxidase. This reflects the

marked enhancement by ethylene of the rate of reaction of superoxide anion radical (O_2^-) with peroxidase[α] (Smith et al. 1982, 1983) (cf. the action of ethylene with another heme protein, cytochrome P-450 in animals, described below). Ethylene also influences ion and sucrose flux in plants (Hanson and Kende 1975; Eastwell and Spencer 1982b). Eastwell and Spencer (1982b) showed that the release of calcium ions (Ca^{2+}) was much more sensitive to ethylene than the release of any of the other ions investigated. Ethylene (13.1 $nL \cdot mL^{-1}$ (ppm) of purified air) induced a 101% increase in the release of Ca^{2+} from gibberellic acid - treated aleurone layers. This effect of ethylene may be of considerable significance with respect to calmodulin activity in both plant and animal tissues. These effects may not be confined to plants, but experiments have not yet been conducted with animal tissues.

Recent data suggest that the destruction of cytochrome P-450 plays a role in the hepatotoxicity of ethylene. Cytochrome P-450 is a monooxygenase which, in animals, hydroxylates xenobiotics. This makes them more water-soluble and aids in their excretion and, often, their detoxification. Ortiz de Montellano and Mico (1980) and Ortiz de Montellano et al. (1980, 1981) reported that ethylene, administered as a 1:1 (v/v) mixture of ethylene and air for 3 h, destroys the hepatic microsomal cytochrome P-450 monooxygenase that catalyzes its oxidative metabolism in phenobarbital-pretreated rats. The process results in the N-alkylation of the prosthetic heme of cytochrome P-450, possibly during attempted transfer of the catalytically activated oxygen to the π-bond of ethylene, to form an abnormal hepatic porphyrin. The ethylene adduct was identified as N-(2-hydroxyethyl)protoporphyrin IX (isolated as the dimethyl ester). The alkylation of the cytochrome P-450 heme did not involve intervention of an epoxide metabolite, and was also observed with other olefins, including vinyl chloride. Ortiz de Montellano and Mico (1980) suggested that cytochrome P-450 destruction by endogenous ethylene may be significant under particular conditions of physiological stress. In contrast, Dent and Graichen (1978) failed to demonstrate an interaction between rat liver cytochrome P-450 and ethylene in vitro.

7.3.2 ETHYLENE EXPOSURE FOLLOWING PRETREATMENT

Conolly et al. (1977, 1978) observed hepatotoxic responses in rats pretreated with Aroclor 1254, then exposed via inhalation for 4 h

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[α] Peroxidases are plentiful in higher plants, but rather rare in animals. Leukocytes contain a verdoperoxidase that utilizes a variety of organic substrates as well as a myeloperoxidase that oxidizes halides to halogens, which are bactericidal. Erythrocytes contain glutathione peroxidase, and all heme proteins have a low peroxidase activity (Smith et al. 1983).

to high concentrations of ethylene (11 500, 29 000, 66 000 $mg.m^{-3}$; 10 000, 25 000, 57 000 ppm) or vinyl chloride monomer; both compounds produced severe degeneration and necrosis of the liver, and caused significant elevation of serum alanine-α-ketoglutarate transaminase (SAKT), an indicator of liver injury. These authors interpreted their results as indicating that the toxic injury resulted from induction of the mixed function oxidase system to a level at which the rate of double bond epoxidation exceeded epoxide detoxification.

In another experiment, Conolly and Jaeger (1977) found that liver injury, as measured by levels of SAKT or sorbitol dehydrogenase (SDH), developed progressively over a 24-h period in PCB-pretreated rats exposed via inhalation to high concentrations (11 500, 26 400, 60 800 $mg.m^{-3}$; 10 000, 23 000, 53 000 ppm) of ethylene for 4 h. Overnight fasting, which depleted hepatic glutathione (GSH) of PCB-pretreated rats before exposure to ethylene, significantly increased the hepatotoxicity of ethylene as measured by SDH. The authors suggested that decreased GSH availability for a GSH-dependent detoxification pathway could increase acute toxicity. No effect of trichloropropane epoxide (TCPE) pretreatment on ethylene toxicity was observed; because TCPE inhibits epoxide hydrase (EH), which detoxifies the epoxide, Conolly and Jaeger (1977) expected that if ethylene was metabolized to an epoxide intermediate, TCPE treatment might increase toxicity. However, treatment of fasted PCB-pretreated rats with TCPE before exposure to 11 500 $mg.m^{-3}$ (10 000 ppm) ethylene was synergistic in producing acute hepatic injury.

Conolly and Jaeger (1977) concluded from these results that GSH is more important than EH in the detoxification of toxic chemical species generated through ethylene metabolism, although EH must be at least a minor detoxification pathway. This implies that ethylene oxide is an intermediate in the hepatic metabolism of ethylene in PCB-pretreated rats. The relative importance of EH must increase as GSH is depleted, since fasting alone did not increase acute toxicity as much as fasting plus TCPE pretreatment (Conolly and Jaeger 1977).

Guest et al. (1981) found that rats pretreated with Aroclor, then exposed for 5 h to 11 500 $mg.m^{-3}$ (10 000 ppm) ^{14}C-ethylene, had greatly increased concentrations of radioactivity in the liver, and exhibited centrolobular hepatic necrosis 36 h after exposure, whereas rats exposed to ethylene alone had no light-microscopic evidence of hepatotoxicity. The authors hypothesized that the Aroclor pretreatment, which induces the hepatic mixed function oxidase system, affected the rate or route of ethylene metabolism so that a toxic metabolite was produced; the metabolite was either not produced in the controls or was produced in insufficient quantities to elicit a hepatotoxic response.

The mechanism by which Aroclor potentiates the hepatotoxicity of ethylene has not yet been elucidated. However, because such high doses of both Aroclor and ethylene are required to elicit a toxic

response, Hamm et al. (1984) concluded that results of experiments utilizing Aroclor pretreatment probably have little relevance in assessing potential ethylene toxicity to humans.

7.3.3 ESTIMATES OF RISK

It is apparent from the studies discussed in Sections 7.3.1, 7.3.2, 6.2.2 and 6.2.3 that ethylene may be metabolized in some organisms. It is therefore important to examine not only the effects of ethylene on animals, but also those of its metabolites. Since it has been demonstrated in several organisms that ethylene gives rise in vivo to ethylene oxide, an alkylating agent with established mutagenicity, carcinogenicity and teratogenicity (see, for example, Dunkelberg 1981; Ehrenberg and Hussain 1981; Yager and Benz 1982; Flores 1983; Högstedt et al. 1983; Gennart et al. 1983), and since it is not known whether tissue doses of ethylene oxide generated in vivo from ethylene exert the same effects as those obtained via inhalation of the epoxide, several groups have attempted to estimate the genetic risk associated with ethylene exposure. Ehrenberg et al. (1977) determined the dose of ethylene oxide in mice exposed to ethylene from the degree of alkylation of amino acid residues in hemoglobin in order to estimate the genetic risk to humans associated with exposure to ethylene. A tentative estimate of this risk for a dose of 1.1 $mg.m^{-3}.h$ (1 ppm.h) ethylene was calculated by Ehrenberg et al. (1977) to be about 0.01 mGy-equivalent. (Recent calculations suggest this risk estimate may be too high (Ehrenberg 1985).) For a person exposed to an average ethylene concentration of 0.06 $mg.m^{-3}$ (0.05 ppm), the estimated genetic risk would therefore be ≈0.05 mGy-equivalent per week, assuming a considerable part of the time is spent at rest. The risk from background radiation (≈0.02 mSv per week) is about the same order of magnitude.

Osterman-Golkar and Ehrenberg (1982) performed a similar calculation for rats, and concluded that exposure of rats to 2 $mg.m^{-3}.h$ (1 ppm.h) ethylene oxide (or 49 $mg.m^{-3}.h$ (43 ppm.h) ethylene) leads to a risk of genetic damage corresponding to that of 0.3 mGy-equivalents. These authors state that the resolving power of the hemoglobin dosimetry is about 2 x 10^4 times greater than that of biological test systems for ethylene oxide. Latarjet et al. (1981), who assumed that inhalation of 1.2 $mg.m^{-3}$ (1 ppm) ethylene for 1 h leads to absorption of 20 µg (0.45 µmol) of ethylene oxide, or 0.001 mGy-equivalent, calculated that the maximum acceptable concentration of ethylene in the air for the total population exposed continuously would be 0.23 $mg.m^{-3}$ (0.2 ppm), leading to 0.5 mg ethylene oxide fixed weekly and a total exposure of 1.7 $mGy\text{-equivalents}.a^{-1}$. The authors concluded that present levels of pollution of air by ethylene are in the acceptable range of their recommendations.

This type of calculation on radiation and chemical equivalence has been criticized (e.g. ECETOC 1982; Ehrenberg et al. 1983). Some criticisms are that (i) not all DNA damage is equally liable to develop

into mutational or carcinogenic events, and (ii) mammals have well-developed protective mechanisms which help both to lower tissue doses and to repair damage caused by chemicals, whereas the protective setup against radiation is not as effective (although DNA repair is probably just as efficient) (ECETOC 1982).

Calleman (1982) calculated the *in vivo* doses of ethylene oxide in rats exposed to ethylene. Since the rate of uptake of ethylene reaches a maximum as the concentration in air is increased (Andersen *et al.* 1980), the resulting *in vivo* dose of ethylene oxide also approaches a maximum. This dose of ethylene oxide is below that which gives a significant response in rats. Calleman (1982) suggested that this may explain why cancer tests with ethylene so far have been negative (e.g. Chemical Industry Institute of Toxicology 1980). Osterman-Golkar and Ehrenberg (1982) also discussed the low statistical power of long-term cancer tests with ethylene.

In general, then, it appears that the hazard to humans and other animals from the inhalation of ethylene and subsequent metabolism to ethylene oxide may be relatively small, judged by traditionally used biological tests, for the following reasons: (i) humans are normally exposed to only very low levels of ethylene in the environment (perhaps 15-70 $\mu g.m^{-3}$ (13-60 ppb), depending on proximity to automobiles and industry; see Section 5.1) (Note that these levels are orders of magnitude lower than those used by most researchers to investigate potential adverse health effects; see Section 7.3.); (ii) much of the inhaled ethylene is rapidly excreted with exhaled breath, the rapid equilibration probably reflecting the fact that ethylene has one of the lowest blood-gas partition coefficients (0.14) of any of the common anesthetic gases (Anon. 1968); (iii) probably less than 10% of the inhaled amount of ethylene is converted to ethylene oxide (see, for example, Segerbäck (1983) (8%) or Latarjet *et al.* (1981) (1%)); and (iv) much of the ethylene oxide formed in the liver by monooxygenase/cytochrome P450 systems is probably detoxified by glutathione transferase enzymes, which are found in the liver at high levels.

An extremely simplistic calculation illustrates these points in a quantitative manner. A rat exposed to 250 $mg.m^{-3}$ (220 ppm) ethylene (a higher concentration than one would normally expect to encounter in the Canadian environment; see Chapter 5) would inhale about 7.5 $mg.kg^{-1}.h^{-1}$, assuming a minute volume of 150 mL and a body weight of 300 g (0.25 $mg.L^{-1}$ x 9 $L.h^{-1}$ x 1000 $g.kg^{-1}$ x 1/300 g = 7.5 $mg.kg^{-1}.h^{-1}$). Assuming 1% of the inhaled amount of ethylene was metabolized to ethylene oxide (this value varies depending on animal species used) (Ehrenberg *et al.* 1977; Latarjet *et al.* 1981), then 0.075 $mg.kg^{-1}.h^{-1}$ of ethylene oxide would be formed. Because Andersen *et al.* (1980) (see Section 6.2.3.1) showed that rats exposed to 250 $mg.m^{-3}$ (220 ppm) ethylene had an uptake of 0.12 $mg.kg^{-1}.h^{-1}$, this means that 63% (0.075/0.12) of the ethylene absorbed by the body is metabolized to

ethylene oxide. Most of this ethylene oxide could then be further metabolized to form additional end products, whereas a small fraction of it could participate in alkylation reactions. This type of calculation would change considerably if the initial concentration of ethylene in the air were changed. For example, at lower concentrations, one would have to consider the possibility that a higher fraction of inhaled ethylene is converted to ethylene oxide (Segerbäck 1983). At higher concentrations, on the other hand, one would have to take into consideration the saturability of ethylene uptake, the maximum rate being 0.24 $mg.kg^{-1}.h^{-1}$ (Andersen et al. 1980).

Filser and Bolt (1984a) and Bolt and Filser (1984) concluded that inhalatory exposure of rats to 1150 $mg.m^{-3}$ (1000 ppm) ethylene or more would theoretically correspond to about 14 $mg.m^{-3}$ (7.5 ppm) ethylene oxide exposure, taking into consideration the distribution pattern of ethylene oxide in rats exposed to high levels of ethylene and the saturation behavior of ethylene metabolism. At exposures below 1150 $mg.m^{-3}$ (1000 ppm) ethylene, the equivalent ethylene oxide concentration would theoretically be 1/133 that of ethylene, because of ethylene's first-order metabolism in this range (Bolt and Filser 1984); the data of Ehrenberg et al. (1977) and Segerbäck (1983), on the other hand, are compatible with Michaelis-Menten kinetics at low concentrations of ethylene. Bolt and Filser (1984) suggested that a threshold limit value (TLV) of 1.8 $mg.m^{-3}$ (1 ppm) for ethylene oxide would be in accordance with a TLV of 115 $mg.m^{-3}$ (100 ppm) for ethylene.

The Interdisciplinary Panel on Carcinogenicity (1984), convened at the request of the American Industrial Health Council, recently reviewed and reevaluated criteria for assessment of evidence of carcinogenicity of chemicals. It states: "Toxicological evaluation of carcinogenicity has classically relied upon long-term in vivo studies as the primary source of data. Such studies have been performed in a routine manner, and evaluations have followed pre-determined formulas. This rote method is rapidly giving way to evaluations that take into account findings from in vitro tests, metabolism studies, and biometric analyses as well as any other available information. One of these methods alone cannot provide a reliable estimate of a chemical's risk to man, but taken together they provide an estimate with a high level of confidence." It concludes that "the characterization of human risk always requires interdisciplinary evaluation of the entire array of data on a case-by-case basis. Animal studies, whenever possible, should be augmented by studies of mechanism, metabolism, and pharmacodynamics."

8.0 AIR QUALITY STANDARDS

At present, there are no official federal Canadian guidelines for ambient levels of ethylene in air. However, Ontario has a tentative standard of 160 $\mu g.m^{-3}$ (140 ppb) for one half hour based on a 24-h objective of 40 $\mu g.m^{-3}$ (35 ppb) and a 6-h objective of 120 $\mu g.m^{-3}$ (105 ppb) (Linzon 1984). Several standards have, however, been proposed for ethylene in the United States. For example, the California State Board of Health proposed maximum levels of ethylene of 0.6 $mg.m^{-3}$ (0.5 ppm) for 1 h and 0.1 $mg.m^{-3}$ (0.1 ppm) for 8 h (California State Board of Health 1962, cited by Abeles 1973). The American Industrial Hygiene Association (Anon. 1968) suggested the maximum levels of ethylene in air shown in Table 8-1, with the caveat that there was insufficient knowledge on which to base an unequivocal ambient air standard for the gas. Abeles (1973) feels these maxima are 10 times too high.

In the absence of sufficient data on the possible effects of ethylene on organisms in the natural environment, it is difficult to make suggestions that might aid in the formulation of air quality standards for ethylene in Canada. It is clear, however, that a typical threshold level for certain physiological effects of ethylene on vegetation in laboratory conditions is around 0.01 $mg.m^{-3}$ (ppm) for a few hours of exposure. This creates a rather odd situation regarding the establishment of air quality standards since even in a rural unpolluted environment one can commonly find this concentration of ethylene. In cities the levels may on occasion greatly exceed this.

This situation is further complicated by the possibility that the sensitivity of organisms to ethylene may vary with time of day, time of year, and stage of development.

Thus, attempts should be made not to greatly exceed the levels of ethylene that might normally be found in an unpolluted rural environment. If these levels are exceeded, the duration of the event should be kept as short as possible.

Table 8-1. Recommended levels of ethylene in air (from Anon. 1968).

Area	1-h maximum		8-h maximum	
	$mg.m^{-3}$	ppm	$mg.m^{-3}$	ppm
Rural	0.29	0.25	0.058	0.05
Residential	0.58	0.50	0.12	0.1
Commercial	0.86	0.75	0.17	0.15
Industrial	1.15	1.00	0.23	0.20

LIST OF ABBREVIATIONS AND TERMS

AB	abscisic acid
ACC	1-aminocyclopropane-1-carboxylic acid
Aroclor	a mixture of polychlorinated biphenyls (tradename)
ATP	adenosine triphosphate
ATPase	adenosine triphosphatase
CoA	coenzyme A
DNA	deoxyribonucleic acid
EF	emission factor(s)
Eh	redox potential
EH	epoxide hydrase
Et	ethylene
Ethephon	$Cl\text{-}CH_2\text{-}CH_2\text{-}PO_3H_2$, a chemical that liberates ethylene
FID	flame ionization detection
GA	gibberellins
GC	gas chromatography
GSH	glutathione
Gy-equivalent	unit equating the dose of a chemical agent producing a given genetic effect to the dose of ionizing radiation producing the same quantitative effect for the same genetic end point (see Latarjet *et al.* 1981 for detailed explanation)
Hb	hemoglobin
KGA	α-ketoglutarate
K_m	Michaelis-Menten constant, the substrate concentration at which the velocity is half maximal
KTBA	2-keto-4-thiomethylbutyric acid

LIST OF ABBREVIATIONS AND TERMS (Cont'd)

MTA	5'-methylthioadenosine
MTR	5-methylthioribose
NADPH	nicotinamide adenine dinucleotide phosphate (reduced)
OD	outer diameter
PID	photoionization detection
PCB	polychlorinated biphenyl
RNA	ribonucleic acid
s	second
SAKT	serum alanine-α-ketoglutarate transaminase
SAM	S-adenosylmethionine
SDH	sorbitol dehydrogenase
Sv	sievert, a unit of dose equivalent (1 Sv = 100 rem (rad-equivalent man))
TCA	tricarboxylic acid
TCPE	trichloropropane epoxide
V_{max}	maximum velocity
VOC	volatile organic compound(s)

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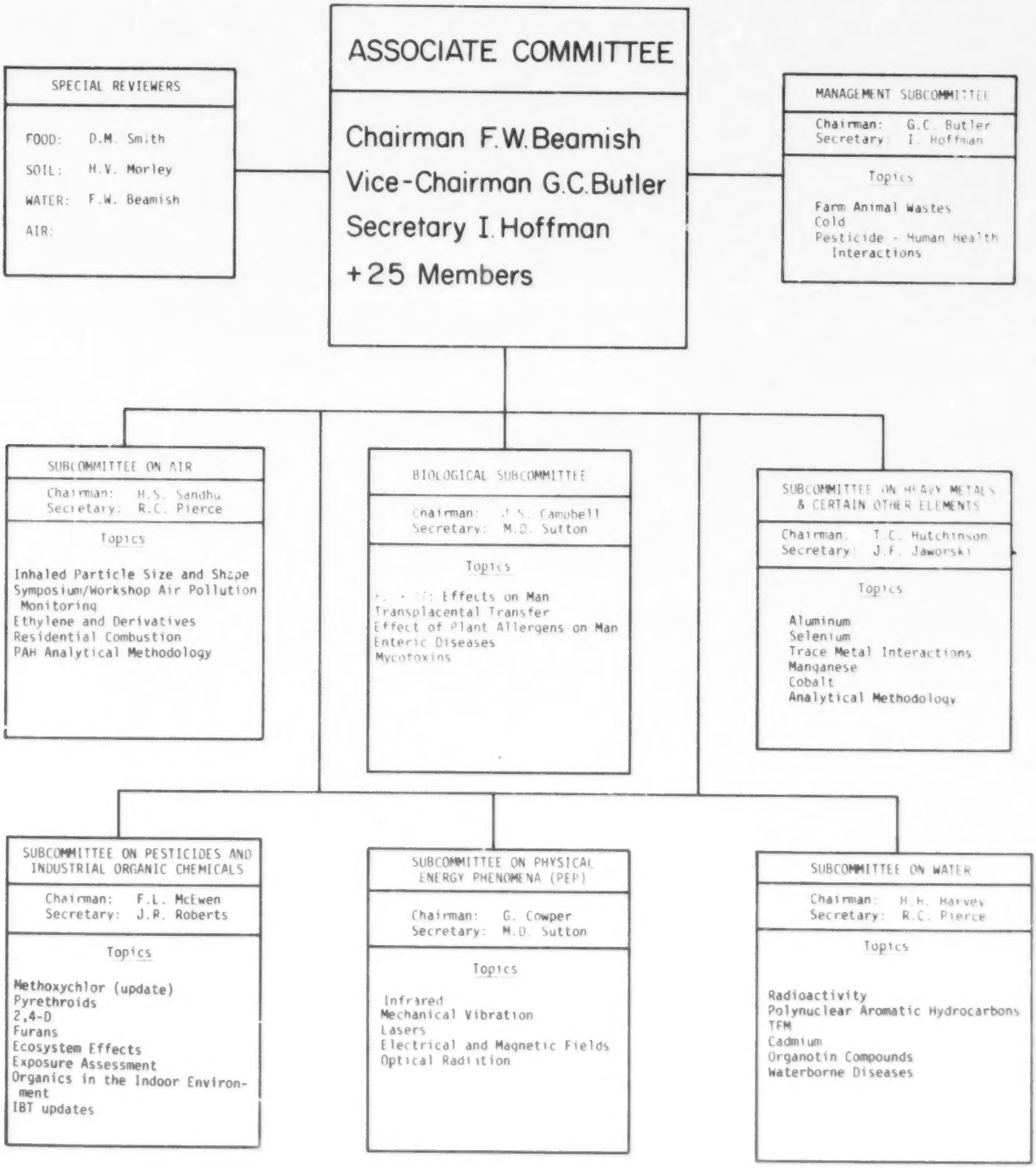
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